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THE LUMLEIAN LECTURES

FOR 1900

ON SOME
CIRRHOSES OF THE LIVER

BEING THE

LUMLEIAN LECTURES FOR THE YEAR 1900

DELIVERED BEFORE THE

ROYAL COLLEGE OF PHYSICIANS, LONDON

BY

WALTER BUTLER CHEADLE, M.A., M.D. CANTAB.

FELLOW OF THE COLLEGE; SENIOR PHYSICIAN AND LECTURER ON
CLINICAL MEDICINE, ST MARY'S HOSPITAL
CONSULTING PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND ST.

WITH ILLUSTRATIONS

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PREFACE

In publishing these Lectures I must thank those of my colleagues who have kindly permitted me to make use of cases under their care, and express my obligation to my friend, Dr. Poynton, for much valuable assistance in many ways, and more particularly in the preparation of specimens in illustration of the subject, for which I am largely indebted to him.

19 Portman Street, London, W.
May 1900.

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ON SOME

CIRRHOSES OF THE LIVER

LECTURE I

Introductory remarks—Object of the lectures—Diseases of the liver—Difficulties which attend their investigation—The three chief forms of cirrhosis—Atrophic, hypertrophic, syphilitic—Cardiac cirrhosis—Undetermined questions—Factors in causation—Atrophic cirrhosis—Enlargement in the early stage—Ascites—Jaundice—Viability—Character of the fibrosis—Association with Bright's disease—Hypertrophic cirrhosis—Character of the fibrosis—Incidence of jaundice—Ascites in the hypertrophic form—Pain and pyrexia—Typhoid state—Chronic course—Hanot's biliary cirrhosis not representative—Syphilitic cirrhosis—Character of the fibrosis—Perihepatitis a marked feature—The cirrhosis of congenital syphilis.

MR. PRESIDENT AND GENTLEMEN,—When I was invited to give these lectures I felt very sensible of the honour conferred upon me. As soon, however, as the glamour of the situation moderated, and the prospect cleared, I realised how difficult it would be worthily to fulfil the duty laid upon me. I called to mind the ox and the charger in the fable—'optat ephippia bos piger, optat arare caballus.'

I reflected, however, that there is always something of value to be learned from the honest observer, and in this belief I venture to offer to you some results of my clinical experience.

In these later times that important organ the liver has, I think, hardly received the attention which it deserves.

The rapid development of our knowledge of the nervous system and its diseases has largely absorbed the interest of clinical observers; and the apotheosis of the bacterium and the bacillus have caused those organs which do not favour their exploitation to be somewhat neglected.

The heart indeed has held its own. It has a natural attractiveness—even fascination—for the clinical observer. The mechanism is well understood, the condition of its machinery ascertained with comparative ease and precision by various simple means.

The lungs also in like manner are open to physical examination, and are moreover the favourite haunt of micro-organisms; they also retain their position of clinical interest; while the kidneys, although hidden away from direct physical examination, furnish a means of testing their condition through the secretion of urine, which secures their hold upon medical investigation.

The great composite gland the liver, however, is less readily dealt with. Not only are its functions more complex and varied than those of any other abdominal organ, but it is a still and silent worker, doing its office noiselessly and without movement.

There is no portion of the digestive machinery whose integrity is more important to nutrition; none in which slight disturbances are more common, or more productive of discomfort and impaired health ; and yet there is no one of the viscera whose aberrations of function are less accurately and scientifically ascertained.

The physical condition of the liver can only be examined during life by the sense of touch, showing its size, contour, and consistence, aided in minor degree by auscultation and percussion. The state of its complex functions can only be inferred—and that imperfectly —by the effects of their disorder upon other organs.

The difficulties which beset the investigation of condition of the liver and its functions during life have prevented full knowledge being gained on certain points in its pathology, and the clinical history of its diseases has remained in many respects unsettled.

Much excellent work has been done by many physicians during the last twenty years, but much remains to be done.

Amongst undetermined questions with regard to disorders of the liver—questions which still remain the subject of discussion, and about which opinion is not yet finally formed—are some relating to the cirrhoses ; and it will be my endeavour in these lectures to throw light upon certain of the obscurities which still beset this group of hepatic affections.

During the last sixteen years, the period for which my attention has been specially directed to this subject of hepatic cirrhoses, a series of cases have come under my observation which have not corresponded strictly or satisfactorily in clinical or pathological characters with the description of the disease in its different forms as set forth by authority. I believe that the views generally regarded as established require modification and expansion.

The lines laid down are altogether too absolute and too rigid. The conclusions drawn at first, and accepted with little or no question, are necessarily built upon imperfect knowledge, and require revision from time to time as experience grows.

The clinical picture of cirrhosis, for example, gloomy as is the reality in many respects, has been painted in more melancholy colours than is altogether warranted by facts.

The interest in the disease has centred in the post-mortem room rather than at the bedside; and the revelations of the former have overshadowed the brighter possibilities which are sometimes indicated by the latter. In passing judgment upon the disease all forms of it have commonly been vaguely run together, under the general term *cirrhosis*, and the last stage of the most fatal condition has been regarded as representative: the remediable and the irremediable have been confused together in one stereotyped picture of unalterable disease; and this absolutely unfavourable verdict has, I think, been largely influenced by the disastrous results of mistaken treatment.

Cirrhosis of the liver occurs in various forms and in different degrees. It does not, as some would appear to conceive, arise suddenly, fully developed, in the extreme form of the final stage of atrophic degeneration.

There are cirrhoses and cirrhoses, and each has its successive stages of development. Cirrhosis is no doubt a subtle and secret malady; as a rule its existence is not suspected until it has reached such degree that grave symptoms have arisen to draw attention to its presence.

But disease does not always proceed upon the lines laid down for it; is not always rigidly cut to pattern, bound by routine and red tape; it is occasionally unorthodox; and cirrhosis now and again betrays itself at an earlier period of its course than the final stage prescribed for its disclosure. Symptoms such as ascites may anticipate the ordinary term and give opportunity for remedial treatment, so that in comparatively favourable, although infrequent, cases the patient may recover for a time of varying length —months or even years—and be afforded a respite of appreciable value.

It is my purpose to deal mainly with the three chief forms of cirrhosis, and those with which ascites in its most pronounced and characteristic form is especially associated. It is on those distinguished as *atrophic cirrhosis*, *hypertrophic cirrhosis*, *syphilitic cirrhosis*, that the main interest of the subject centres.

Before proceeding further, however, it will be well to clear the ground by putting aside certain minor forms which lie outside the strict lines of the present inquiry. Amongst these are those conditions of fibrosis which are due to the obstruction of venous escape from the hepatic vein—as in mediastinal tumours, mediastinitis—or associated with general dropsy in mitral and tricuspid disease of the heart. The hepatic fibrosis which is set up by the chronic venous congestion of this kind is, as a rule, a slight cirrhosis only, not usually sufficient in itself to interfere materially with nutrition, or to cause such serious portal obstruction as to give rise to abdominal dropsy. The peritoneal effusion is not great, and forms merely a part, as it were, of the general dropsy which follows the damming back of blood in the systemic venous system.

Yet in some instances of this congestive cirrhosis we meet with an ascites as a distinct feature which is out of all proportion to the effusion elsewhere; not merely a part of general dropsy, but a special effusion into the peritoneal cavity: a true ascites, due to added difficulty in the portal area; the systemic obstruction, insufficient alone to produce a general dropsy, reinforced by the *hepatic* obstruction, causes *peritoneal* dropsy. I wish to draw attention to this because it has, I think, an important bearing upon certain cases of hypertrophic cirrhosis of which I have to speak later. The condition of pure cardiac cirrhosis considerable enough to produce ascites is shown in the drawings given at pp. 103 and 104 (Plates X. and XI.), from a case under the care of Dr. Penrose, to whom I am indebted for permission to make use of it.

The great trio of representative hepatic fibroses which have been distinguished as *atrophic cirrhosis*, *hypertrophic cirrhosis*, *syphilitic cirrhosis*, all marked by the increased formation of fibrous tissue in the structure of the liver, in their perfect type differ in clinical character, course, and pathological anatomy each from the other.

Although these things are familiar to all you will pardon me perhaps if, for the sake of greater clearness, I enumerate briefly the special characters which have been generally regarded as distinctive of each of the three cirrhoses.

THE THREE CHIEF CIRRHOSES

1. In atrophic cirrhosis the fibrous invasion is described as spreading along the main branches of the interstitial tree, embracing groups of lobules and compressing them, and thus causing atrophy and destruction of cells.

The new fibrous growth between the inclosed groups of lobules contracts, and drawing in the surface at these points causes hobnailing of the surface and shrinkage of the whole organ, so that it is smaller than normal, decreased in weight, tough, and hard.

Amongst the clinical features which are laid down as characteristic of atrophic cirrhosis, as contrasted with other forms, are signs of portal obstruction, evidenced by the frequency of ascites, and the occurrence of hæmorrhage from the stomach and bowels; the rarity of jaundice; the short course of the disease after it can be recognised; its inevitable fatality; and that it is almost invariably set up by chronic alcoholic irritation—*atrophic multilobular cirrhosis, or hobnail liver.*

2. In the second form of cirrhosis, the hypertrophic form, described by Hanot as 'biliary cirrhosis,' the liver condition is stated to be characterised by increase in size and weight: the fibrosis which invades the interstitial tree affects the smaller twigs rather than the main branches, embracing single lobules, and even single cells, instead of groups of lobules; accompanied, moreover, by a curious development of biliary canaliculi at the periphery of the lobule, due either to the formation of new ductules there, or to the exposure of existing ones by denudation through the destruction of hepatic cells. In this form the liver is greatly enlarged: it is hard, smooth of surface, or only slightly granular—*hypertrophic monolobular biliary cirrhosis.*

The clinical aspect of hypertrophic cirrhosis as laid down by authority is characterised by features which contrast sharply with those of the atrophic form.

Ascites is described as entirely absent, or, if present, so slight as to be comparatively unimportant; while jaundice, on the contrary, is said to be an almost invariable symptom, and often intense and persistent. Intermittent attacks of pain and pyrexia; enlargement of the spleen; and a liability to the supervention of acute symptoms, such as severe delirium, hæmorrhages, coma, and a chronic course of some years are other signs which distinguish hypertrophic cirrhosis.

Its cause is involved in obscurity; a doubtful factor, in some circumstances supposed to be malarial, in some alcoholic, in some obstruction to ducts, is credited with setting up inflammation of the biliary canaliculi, which is regarded as the starting-point of the pathological change.

3. The third variety of cirrhosis—the syphilitic—is described as characterised by the presence of broad bands of fibrous tissue, separating isolated masses of liver tissues and causing destruction of hepatic cells.

The organ is often much deformed by the gradual contraction of these several fibrous bands, giving it a lobulated appearance, its surface marked by rounded bosses, often scarred and furrowed, or only slightly uneven. Gummata may also be present in various stages.

A hard liver; usually enlarged; sometimes contracted, with uneven, bossed, or slightly irregular surface.

The clinical features of this kind of cirrhosis closely resemble one or other of the preceding forms—in the early stage of enlargement the hypertrophic, in the later either the hypertrophic or atrophic alcoholic form. In one case jaundice, no ascites; in another both; in a third neither, but, as a rule, when advanced, ascites and perihepatitis.

There is another form of syphilitic cirrhosis described as chiefly limited to the hereditary syphilis of childhood—a diffuse fibrosis—characterised microscopically by its immature partly cellular character penetrating the lobules, and even between the cells. This is shown in Plate VII. (*a*), given at page 45.

The liver is greatly enlarged; clinically jaundice, ascites, and emaciation are amongst the chief symptoms.

POINTS IN THE PATHOLOGY AND CLINICAL HISTORY OF THE CIRRHOSES NOT YET FINALLY DECIDED

Although there is a tolerably consistent general agreement as to the main features of the morbid anatomy of cirrhosis, there is still some confusion as to the relation of the different changes to clinical conditions; discrepancies of opinion on certain points of ætiology; divergent views as to the symptoms and course of the disease in its different forms.

QUESTIONS AWAITING FINAL SETTLEMENT

Amongst the questions awaiting an accepted and final settlement are the following:

Is atrophic cirrhosis—*i.e.* the contracted hobnail liver—always of alcoholic origin? Is the liver small from the first? Or is there an earlier stage in which it is enlarged, becoming contracted later? Or is atrophic cirrhosis merely a late stage of hypertrophic cirrhosis?

Or is there an alcoholic cirrhosis in which the liver is hypertrophied and enlarged from first to last? Or is hypertrophic cirrhosis limited to the biliary cirrhosis of Hanot and Charcot of uncertain origin? Or, it is even asked, does the biliary form described by French authorities exist at all as a distinct entity?

Again, is ascites practically limited to the atrophic form, almost unknown in the hypertrophic? While on the other hand, is jaundice rare in atrophic cirrhosis? —almost invariable in the hypertrophic form?

Are the microscopic characters of monolobular cirrhosis, with hyperplasia of the bile-ducts, as described in the biliary hypertrophic cirrhosis of Hanot and Charcot, found only in cases which correspond to that clinical type? Or are they met with in both atrophic and hypertrophic cirrhosis of alcoholic origin?

The part played by alcohol, syphilis, and malaria in causation and their connection with special types of affection; the incidence and significance of perihepatitis; the curability of ascites; the efficacy of paracentesis, or its uselessness, in the treatment of it; the possibility, not of curing cirrhosis, but of prolonging the life of the cirrhotic patient by this and other means; the conditions under which the course of cirrhosis can be favourably influenced in this way, are other points about which there is a lack of general agreement.

It is to some of these questions that I shall endeavour to find an answer.

ATROPHIC CIRRHOSIS: FACTORS IN CAUSATION

Atrophic cirrhosis of the contracted hobnailed liver would appear to be almost invariably of alcoholic origin. It is said that malaria sometimes plays a part in its production, but the evidence of this is by no means conclusive. I have met with no instance of the kind; yet it is not intrinsically improbable that a disease like malarial fever, which sets up such marked fibrosis of the spleen, should also set up an analogous fibrosis of the liver. In those cases attributed to malarious influences, however, when the cirrhosis is of the contracted hobnailed variety, it is probable that alcohol, an agent by no means inactive in malarial districts, is largely concerned in its production.

THE QUESTION OF ENLARGEMENT IN THE EARLY STAGE OF ATROPHIC CIRRHOSIS

Another question which has been much debated is, 'Is the liver enlarged in the early stage of atrophic cirrhosis?' Is it first hypertrophied, and subsequently diminished in size as fibrotic contraction proceeds? The older writers generally held that this was so occasionally, but that it was exceptional. Murchison[1] expressed the opinion that it was a matter for further investigation whether, if a patient with enlarged liver survived long enough, the enlargement would be followed by cirrhotic contraction.

In one case reported by him in which the liver was greatly enlarged from cirrhosis attributed to alcohol and malaria, the organ was found to be of its natural size three years later.

The patient was treated with mercury, however, and it is doubtful if the condition was one of simple alcoholic cirrhosis. The patient got well and the decrease of size ceased at the normal level.

Instances have been recorded by Dr. Frederick

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[1] *Dis. of Liver*, 2nd ed. p. 140.

Taylor[1] and others in which the greatly enlarged liver of alcoholic cirrhosis has been observed to undergo contraction. I have watched a number of these cases of cirrhosis with enlargement for considerable periods—one for nearly thirteen years, one for eight years, one for two years. In the first there has been some slight decrease in size, but great enlargement is a characteristic feature still, and in no case have I observed the condition change from an enlarged to a small contracted liver.

I think, however, that there *is* a stage of engorgement and congestive swelling in the atrophic cases before fibrotic contraction is fairly in process.

We are all of us familiar with the enlarged liver of the drinker, who has not developed any symptoms of actual cirrhosis. This, however, is quite distinct from the large hard liver of hypertrophic cirrhosis, which does not merge into the atrophic form. Such contraction as that described in Dr. Taylor's case must at least be extremely rare. The hypertrophied liver does not represent the early stage of the contracted form. The microscopic characters alone negative this—in the one the fibrosis multilobular almost invariably, in the other monolobular or a combination of the two. Hanot, indeed, recognised that hypertrophic cirrhosis of alcoholic origin was not merely an early stage of the atrophic cirrhosis of Laennec.[2]

The error of regarding the hypertrophic and atrophic condition as different stages of the same thing has

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[1] *Guy's Hospital Reports*, 1888, vol. xlv. p. 310.

[2] Hanot and Gilbert, *Bull. Soc. Méd. des Hôpitaux*, 1890, vol. clxvi.

PLATE I

SIMPLE PERILOBULAR ATROPHIC CIRRHOSIS OF THE LIVER, WITH MUCH FATTY CHANGE, DUE TO ALCOHOLISM

The patient, J. K., aged 54, had been a heavy drinker. He was admitted for ascites, and was tapped three times. Finally he became delirious, and died after a terminal hæmatemesis.

Post-mortem.

The *liver* weighed 41 oz. and was extremely hobnailed.

The *spleen* weighed 15 oz.

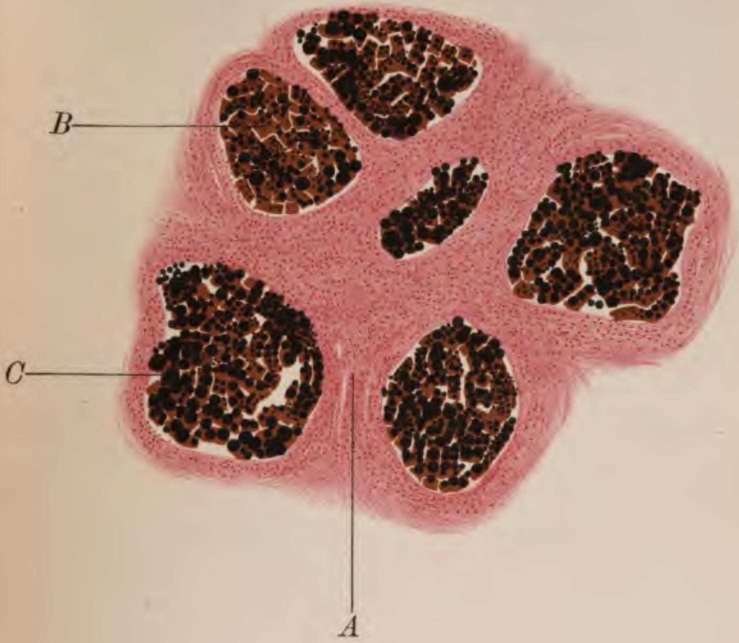
The *pancreas* and *kidneys* showed some fibrosis.

The *heart* was fatty. There was no chronic peritonitis.

Explanation of the Plate.

A. Coarse cirrhosis.
B. Liver lobule.
C. Fatty change.

Fixative, Herrmann's fluid (osmic).
Stain, Safranin.



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probably arisen from its being observed that in some cases the liver was large, in others small; and since a permanent hypertrophy was supposed to be limited to Hanot's biliary form, the inference was too hastily drawn that in alcoholic cirrhosis the large and small were connected, and merely different stages of the same affection. They are distinct varieties, rather than different stages of alcoholic cirrhosis.

ASCITES IN ATROPHIC CIRRHOSIS: ITS SIGNIFICANCE. VIABILITY IN ATROPHIC CIRRHOSIS

In the next place, is the occurrence of ascites a special feature of atrophic cirrhosis, and practically limited to the final stage of it? And is it as rare in the hypertrophic form as it is common in the atrophic?

There can be no doubt as to the frequency of ascites in atrophic cirrhosis. In twenty-three consecutive cases of fatal cirrhosis at St. Mary's, in which the liver was small (under 60 oz.), ascites was present in seventeen, absent in six only.

Nor can there be any question of its grave significance in the majority of cases. It indicates, generally, that the disease has progressed towards its end; that the collateral circulation established has become inadequate to the full relief of the obstructed portal circulation; and that the destruction of liver tissue, advancing usually *pari passu*, has reached the limit of viability.

As a rule, but by no means invariably, the patient has only a few months to live; now and again, however, the months are extended to a year or two. As a matter of fact, the supervention of marked jaundice in atrophic cirrhosis is of more immediately fatal augury than the appearance of ascites. There are, however, cases of atrophic cirrhosis in which ascites supervenes before the final stage, when sufficient glandular structure still remains intact, and functionally effective, to carry on life ; and the obstruction to the portal system is not yet so great as to be past compensation by collateral channels, so that partial recovery may take place for a time.

The stage of the liver change at which such survival for a time, after the advent of ascites, is possible, is illustrated by the case of a woman (S. D.) admitted to St. Mary's for hæmatemesis and ascites in October 1891. She was a confirmed spirit drinker. She was tapped three times at intervals, and recovered her general health sufficiently to return to her usual home life, but came into hospital more than a year later with acute bronchitis, of which she died.

Post mortem, the liver was found to be moderately hobnailed, small, but microscopically a large portion of liver tissue, calculated at about one-half, was found to be normal, and presumably effective. Her condition, although vulnerable, had been still viable, under favourable circumstances.

I might adduce a number of other examples, which show in like manner that the occurrence of ascites in atrophic cirrhosis is not always such sentence of immediate death as is commonly inferred. This evidence will, however, be brought forward more appropriately when I come to speak of prognosis.

THE OCCURRENCE OF JAUNDICE IN ATROPHIC CIRRHOSIS

In the standard descriptions of the symptoms pertaining to atrophic cirrhosis a point is made of the fact that although a slight icteric tint may be present marked jaundice is conspicuously absent, as compared with its almost invariable presence in the hypertrophic form.

Here, again, we have been misled by the assumption that hypertrophic cirrhosis usually corresponds with Hanot's special biliary form.

The matter is of no great moment, but no such hard-and-fast line as to the occurrence of jaundice can be drawn. Jaundice of a marked character is not consistently absent in the small liver of alcoholism, and on the other hand it is by no means constantly present in the hypertrophic form. Murchison[1] observed that, although decided jaundice is rarely met with in atrophic cirrhosis in the early stage, it may occur in advanced stages, and is then an ominous symptom. Dr. Foxwell,[2] as the result of his investigation of sixty-seven cases, concludes that jaundice and cerebral symptoms are as common when the liver is small as when it is large. In the fifty-three fatal cases of cirrhosis of all kinds at St. Mary's during the last ten years I find eight in which marked jaundice was observed. In four of these cases the liver was small, and in four enlarged.

The distinction between the two forms with regard to the occurrence of jaundice should be, that whilst it is met with pretty equally in both forms it occurs at an earlier stage in the hypertrophic than in the atrophic condition, in which latter it is often indicative of the approach of a fatal termination.

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[1] *Dis. of Liver*, 2nd ed. p. 128.

[2] *Birmingham Med. Review*, vol. i. 1896.

CHARACTER OF THE FIBROSIS IN ATROPHIC CIRRHOSIS

Another question of some interest in connection with atrophic cirrhosis is whether the fibrosis is always of the coarse multilobular kind, affecting the main branches of the interstitial tree, or whether the finer interlobular variety, which is undoubtedly most characteristic of the hypertrophic form, is found in the small contracted liver also. In the fifty-three cases adduced before, the liver was examined microscopically in eighteen. In one only of the distinctly contracted specimens was the fibrosis diffuse, in the rest coarse and multilobular. In a few instances a small amount of finer fibrosis was found invading the lobules.

THE ASSOCIATION OF ATROPHIC CIRRHOSIS AND BRIGHT'S DISEASE

A certain amount of interstitial change is found in the kidneys in a large proportion of cases (about one half) of alcoholic cirrhosis. In most instances it is not extreme, and this fibrosis of the kidney is to be regarded as a result of alcoholism, just as is the fibrosis of the liver, the kidney being affected usually in minor degree. In some cases the interstitial nephritis is advanced, and, associated with the Bright's disease, a special form of cirrhotic atrophy is occasionally found which has been more particularly described by Dr. Fagge and Dr. Hale White. In this the liver is extremely contracted, its substance atrophied, but the connective tissue is not always greatly increased. The capsule is much thickened, and there is also extensive perihepatitis, associated adhesive peritonitis, and ascites which is attributed chiefly to the perihepatitis and chronic peritonitis.

This condition of perihepatitis and chronic peritonitis is well exemplified by a case that was under my care in 1896.

M. H., a woman of fifty, had been a patient at intervals for the previous twelve months for ascites, and had been tapped nine times. The last operation proved fatal owing to septic peritonitis. The stomach and intestines were thickened and adherent from old peritoneal inflammation, of which the hepatic region was the chief centre. The liver was small, surrounded by a thick false membrane, moderately cirrhosed, weighing 40 oz. The kidneys were extremely granular.

This same condition of perihepatitis and dry peritonitis with adhesions occurs, however, in atrophic cirrhosis apart from Bright's disease. In one case (that of J. T., December 2, 1898, who was tapped eleven times) perihepatitis, chronic peritoneal adhesions, perisplenitis, thickening of the intestines, were found in conjunction with advanced cirrhosis, the liver weighing only 33 oz., but the kidneys were normal.

In another still more extreme case under my care—that of a woman of 39 (E. M.), a confirmed gin drinker—this condition of perihepatitis and associated adhesive peritonitis was equally marked. She was admitted first in July 1893 for ascites and peripheral neuritis, tapped twice, and discharged relieved; admitted again six months later, for the same trouble, again tapped twice, and discharged relieved; admitted a third time one year later and again tapped. On this occasion, however, suppurative peritonitis followed, apparently from fouling of the drainage tube, which was accidentally displaced during the process, and the case ended fatally. Post-mortem examination revealed extreme perihepatitis, with thickening and adhesion of adjacent intestines, advanced cirrhosis of the liver, which was typically hobnailed and weighed only 18 oz. The kidneys were not affected.

In the fifty-three cases mentioned there are fifteen in which extensive perihepatitis is recorded, usually with extension of peritonitis to neighbouring structures, and adhesions, notably of the diaphragm, stomach, and transverse colon.

Of these fifteen four were syphilitic; in one of these parenchymatous nephritis was present, in the remaining three the kidneys were normal. Of the eleven others in four the kidneys were more or less granular, in three enlarged, and in four normal.

In twenty-two cases the perihepatitis was slight or not recorded.

In four cases there was acute peritonitis. Of these the kidneys were fibrosed in three, in one (which was syphilitic) there was cloudy swelling.

In five cases of the fifty-three there was some chronic peritonitis other than perihepatitis. Of these in one the kidneys were granular, in two large and tough, and in two not affected.

In six of the last cases examined, in which the condition was specially noted, there was no peritonitis. In five of these the kidneys were cirrhotic, in one congested only.

Since, therefore, in those cases of cirrhosis with extensive perihepatitis and adhesive peritonitis of neighbouring structures the kidneys are not constantly affected, and on the other hand when the kidneys are granular there may be no peritonitis, there is no necessary dependence of one upon the other, and the presumption is that the cirrhosis, perihepatitis, and Bright's disease are probably due to the same cause—chronic alcoholic poisoning.

As I shall show later, the most marked instances of perihepatitis and related peritonitis which I have seen have been in connection with syphilitic cirrhosis, in which connection Murchison also observed it.

HYPERTROPHIC CIRRHOSIS

The first question which arises with respect to cirrhosis with enlargement of the liver is whether the affection described by Hanot and Charcot as a biliary cirrhosis of uncertain origin is to be accepted as the representative form of hypertrophic cirrhosis; or if it exists even as a separate entity, or is merely a modified form of alcoholic cirrhosis.

It will be remembered that hypertrophic cirrhosis is described as having a special morbid anatomy and a distinct clinical history and course, which give it a marked individuality of its own.

I think we have been unduly biassed by the classical description of Hanot, as we were formerly by Laennec's description of the atrophic form, and have had no eyes to see obvious facts. Just as deference to Laennec's authority led to the too ready assumption that an enlarged fibrosed liver must be an early stage of the atrophic form, so when the existence of a distinct hypertrophic condition, always hypertrophic and of special character, was demonstrated, it was assumed that every cirrhosis with enlargement must conform to this special variety.

It is surprising how a definite observation of this kind, stated with authority, holds sway, and its general application remains unquestioned, as all comprehensive, without further examination or inquiry.

Subsequent research, however, has proved that many of the features which were considered to be distinctive of the so-called biliary cirrhosis are not peculiar to that exact type, but occur also in cirrhoses which in other points do not conform to Hanot's description.

THE CHARACTER OF THE FIBROSIS

Thus, for example, there is now no question that a fine fibrosis, monolobular or intralobular in arrangement, occurs in conjunction with hypertrophy of the liver in cirrhosis due to alcohol; and this diffuse fibrosis appears also to be met with in the cirrhosis attributed to malaria, which is usually hypertrophic.[1] It is also established, I think without question, that the appearance of hyperplasia of bile ducts in the highly nucleated tissue between the lobules is not met with in cirrhosis with the clinical characters of Hanot's form alone.

Dr. Saundby[2] has shown that this appearance of new canaliculi occurs in cases of ordinary cirrhosis, and also that it is not always present in any marked degree in cases presenting the clinical characters of biliary cirrhosis.

Dr. Frederick Taylor has produced evidence of a like nature.[3] In a case of alcoholic cirrhosis with enlarged liver recently under my care this appearance was found in conjunction with a coarse fibrosis; and this same feature of so-called new biliary canaliculi is shown in a case of simple cardiac cirrhosis as in the illustrations (Plates X. and XI., pp. 103, 104); and in another instance a fine fibrosis was observed in a

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[1] Davidson, *Hygiene and Diseases of Warm Climates.*

[2] *Path. Trans.* 1879.

[3] *Guy's Hospital Reports*, 1895, p. 65.

PLATE II

HYPERTROPHIC CIRRHOSIS OF THE LIVER, ALCOHOLIC, SHOWING COARSE AND FINE FIBROSIS

The patient, S. R., aged 45, was admitted for ascites. He was tapped once, and died suddenly five days later.

Post-mortem.

The *liver* weighed 75 oz. and was hobnailed. There was some perihepatitis.

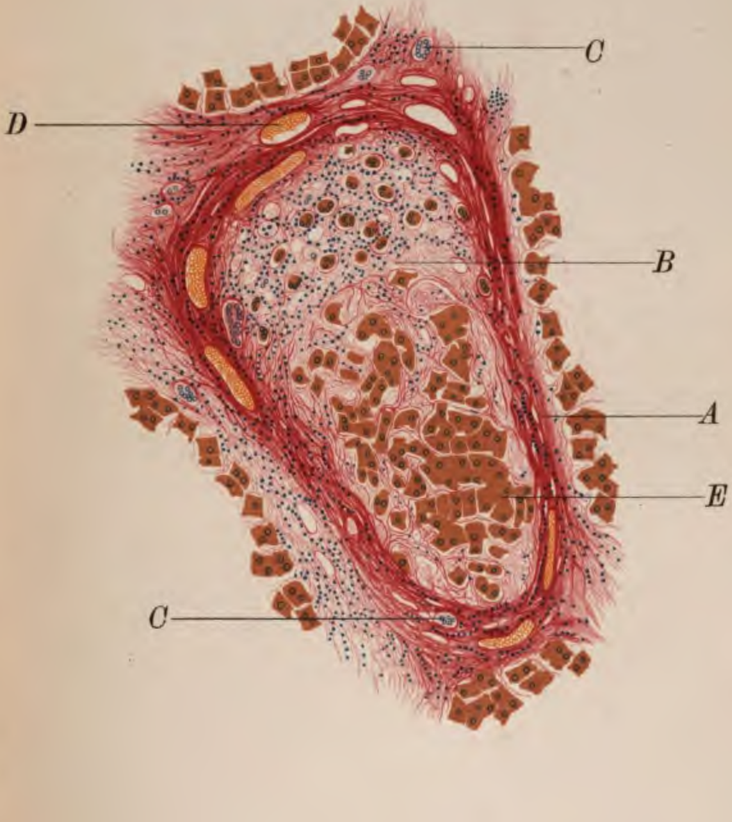
The *spleen* weighed 15 oz. The *kidneys* were distinctly cirrhotic.

Explanation of the Plate.

A. Coarse fibrosis.
B. Fine fibrosis.
C. Pseudo-ducts.
D. Blood-vessels.
E. Liver-cells.

Fixative, Perchloride of mercury.
Stain, Logwood, fuchsin, and orange *g*.

Plate II.



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case of alcoholic cirrhosis without jaundice, in which the liver was small, weighing 53 oz. The interstitial fibrosis most commonly met with in cirrhosis in which the liver is enlarged is a combination of coarse multilobular and fine monolobular development.

The microscopic distinction therefore does not hold.

THE OCCURRENCE OF JAUNDICE

In the next place the presence of jaundice is by no means a constant nor, according to my experience, a very common symptom in hypertrophic cirrhosis.

In seventeen consecutive fatal cases of cirrhosis with enlarged liver, varying in weight from 60 to 134 oz., I find jaundice noted as a marked symptom in four only; in six it is described as slight, and in the remaining seven none is recorded.

Again, out of twenty-seven more cases of cirrhosis with enlarged liver which have either recovered or been relieved, or in which, if fatal, no post-mortem examination was made, jaundice was absent in nineteen, present only in eight.

In ten cases of enlarged fibrous liver from different causes recorded by Murchison, jaundice is mentioned as a symptom in four only; and, as noted before, Dr. Foxwell's investigation of sixty-seven cases of cirrhosis yielded the conclusion that jaundice was as common when the liver was contracted as when it was enlarged.[1]

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[1] *Birmingham Med. Review*, vol. i. 1896.

It is clear, therefore, that jaundice is by no means a constant or characteristic symptom of hypertrophic cirrhosis generally, and, on the other hand, as we have seen before, jaundice in marked degree is by no means rare in the atrophic form.

ASCITES IN THE HYPERTROPHIC FORM

Again, in the typical form of hypertrophic biliary cirrhosis the absence of ascites in any important degree is described as being as distinctive a feature as the presence of jaundice. This certainly does not hold good with regard to hypertrophic cirrhosis which has come under my observation.

In the twenty-five fatal cases of hypertrophic cirrhosis in which the liver weight varied between 60 and 134 oz., ascites was present in sixteen. In six which recovered more or less perfectly, ascites was a prominent symptom in all but one.

In the ten cases of hypertrophic cirrhosis given by Murchison, ascites was present in seven. Bristowe records one of alcoholic cirrhosis with great enlargement, in which the patient was tapped fourteen times for ascites.

In the sixty-seven cases of alcoholic cirrhosis recorded by Dr. Foxwell, he found that ascites was as common when the liver was large as when it was small.

The absence of ascites therefore is not a constant and typical negative symptom of cirrhosis when the liver is enlarged, any more than the presence of jaundice is a reliable sign or distinctive symptom of the hypertrophic form.

It is, I think, probable that this impression of the frequency of the occurrence of ascites in contracted liver and its absence in the enlarged liver has arisen partly from the fact that atrophic cirrhosis escapes notice until ascites arises to draw attention to it. A liver smaller than natural is not obtrusive.

When the liver is enlarged, however, it challenges recognition, and is often discovered before ascites supervenes.

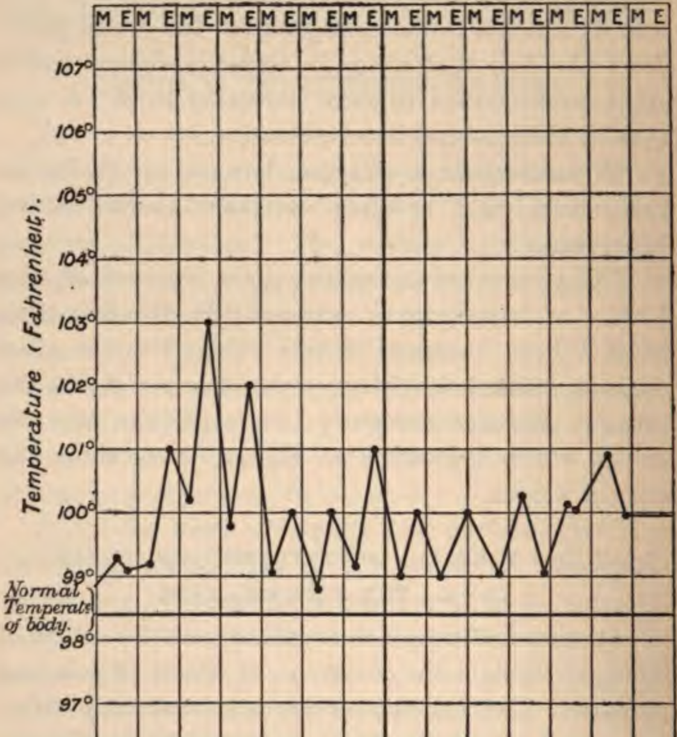
Enlargement of the spleen, again, is not an especial feature of hypertrophic cirrhosis. In the fifty-three cases I have instanced it was enlarged in the great majority of alcoholic cirrhoses, large or small, but the greatest size was reached in the syphilitic cases, in one of which it attained to 42½ oz., or six times the normal weight.

PAIN AND PYREXIA. SUPERVENTION OF DELIRIUM. COMA. THE TYPHOID STATE

Another feature which is said to mark the course of biliary cirrhosis is the occurrence of attacks of pain and pyrexia. These symptoms are not, however, peculiar to cases of the kind. They are met with in atrophic cirrhosis (cases of E. M., 22, 1894; M. D., 10, 1898), in alcoholic hypertrophic cirrhosis without the characteristics of the biliary form, and notably in syphilitic cirrhosis (W. B., 1899).

Pain, tenderness, and pyrexia are, I imagine, merely indications of the supervention of perihepatitis. The charts show the pyrexia, co-existing with pain and tenderness, which mark the presence of perihepatitis in all forms of cirrhosis. Grave symptoms such as delirium, the typhoid state, coma, are not confined to

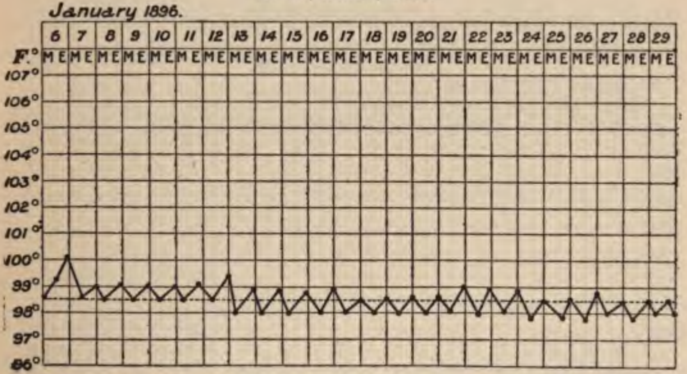
E. M. æt. 38, Jan. 1894.



Atrophic Cirrhosis with Perihepatitis, showing Pyrexial Course of Temperature.

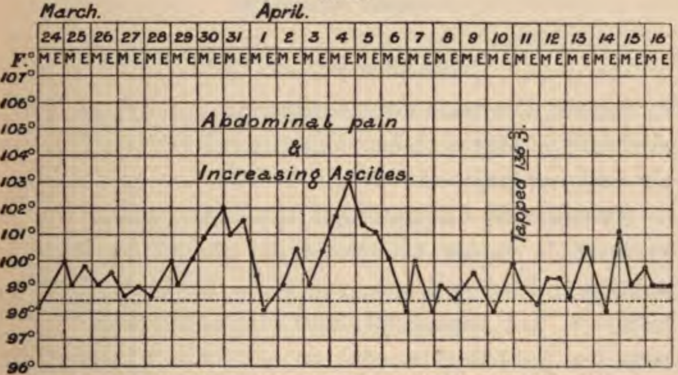
hypertrophic cirrhosis of the so-called biliary type. They occur in hypertrophic cirrhosis of alcoholic origin

R. W., æt. 38, 1896.



Hypertrophic Cirrhosis of the Liver. Chart showing Normal Course of Temperature.

L. P.

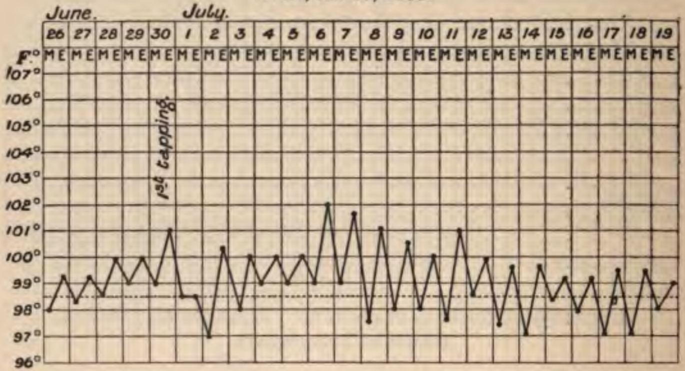


Hypertrophic Cirrhosis and Perihepatitis, showing Pyrexial Course of Temperature.

and type, and, as Murchison noted, and as I have seen (case of extreme alcoholic contracted liver—F.B., 1868), in atrophic cirrhosis also.

These symptoms cannot therefore be regarded as distinctive of a special form of hypertrophic cirrhosis.

W. B., æt. 37, 1899.



Hypertrophic Cirrhosis (syphilitic), showing Pyrexia of extreme Perihepatitis.

THE CHRONIC COURSE

Lastly, the course of the type form of hypertrophic cirrhosis is stated to be a chronic one of two, four, or six years or more.

It must be allowed that the cases which run a protracted course are those in which the liver is enlarged: they are as a rule more benign. This is, however, not invariable, and Dr. Dreschfeld[1] has recently published a case of alcoholic hypertrophic cirrhosis which, while agreeing with Hanot's affection in many respects, differed in the presence of ascites and in the short course of the disease. Chronicity, then, cannot be regarded as a constant character of hypertrophic cirrhosis.

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[1] *Lancet*, Nov. 18, 1899.

It will be seen, therefore, that a cirrhosis in which the liver is greatly enlarged may fail to correspond, in one case or other, in nearly every particular with the classical type, for which we all look, and so rarely find.

Further, from the time of Murchison downwards, a number of cases of cirrhosis have been recorded by Saundby, Bristowe, Dreschfeld, Foxwell, and others in which, although the liver was greatly enlarged, the symptoms have been those usually ascribed to the contracted liver, rather than those of so-called biliary cirrhosis. And in the majority of instances the cause has been traced to alcoholic intoxication.

A number of like cases have come under my own observation, the details of some of which I shall give later. For the present I may say that the hypertrophic cirrhosis I have met with corresponds with the cirrhosis of Hanot chiefly in two respects, viz. in the smooth hard enlargement of the liver, and that the fibrosis is usually diffuse and monolobular, and sometimes partly multilobular. In one instance, however, in which the liver weighed 152 oz., the cirrhosis was coarse and multilobular, with much fatty deposit.

Is it therefore to be inferred that no such special form of hypertrophic cirrhosis as that described by Hanot and recognised by Charcot actually exists? I think we are not warranted in assuming this absolutely.

In addition to the original cases described so graphically by the French writers, other isolated instances have been observed now and again in this country and abroad which agree closely with the so-called biliary type.

For my part I have not, in spite of a close scrutiny for sixteen years, met with a case which has exhibited in full completeness the typical presentment of the French authorities. In three instances only have I seen any close approximation to it.

One of them occurred in a boy of eight (A. A., 1883), who was admitted for jaundice, enlarged liver, and ascites, and died of exhaustion and emaciation four years later. He had had repeated attacks of jaundice since birth. Post mortem a smooth large liver was found weighing 60 oz., with fine diffuse fibrosis, abundant nuclear growth, and imperfectly formed fibrous tissue. There was extensive perihepatitis, and the vessels of the portal fissure were found matted together, the cystic and hepatic ducts being reduced to mere cords, and quite impervious. In another example (E. W., December 1899), in which the patient was admitted for abdominal pain and jaundice, which had existed on and off for six years, the liver was enlarged to the level of the umbilicus, the spleen also enlarged, and ascites was present. Post mortem the liver was slightly granular, and weighed 80 oz. The common bile-duct was found enormously dilated from blockage by two large

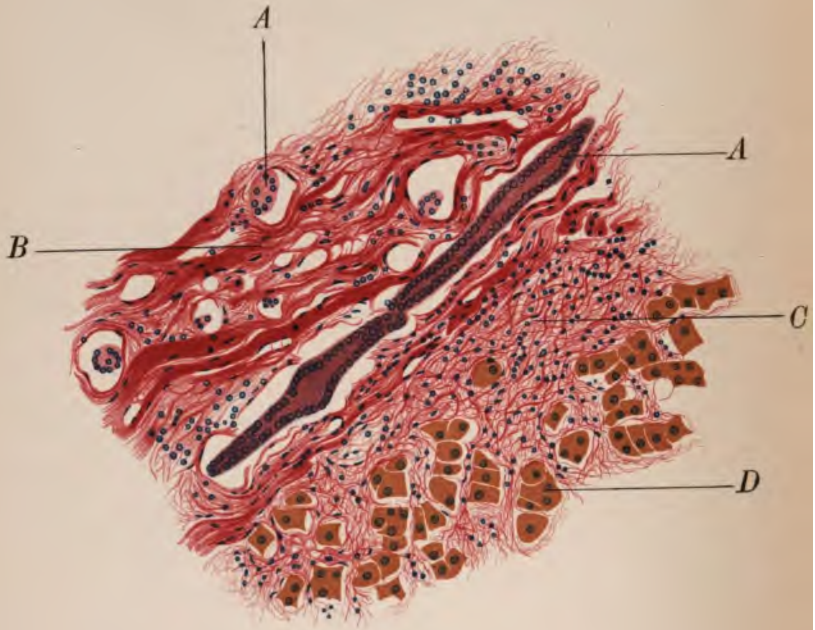

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PLATE III

DIFFUSE CIRRHOSIS OF THE LIVER, SHOWING PSEUDO DUCTS—THE COMMON BILE-DUCT WAS OBSTRUCTED BY TWO LARGE GALL STONES

The patient, E. W., aged 62, had suffered from jaundice for six years.

The liver and spleen were much enlarged, and there was ascites.

He died shortly after admission to the hospital.

Gall stones had been diagnosed four years before.

There was no history of syphilis or alcoholism.

The patient was an army storekeeper.

Post-mortem.

The *liver* weighed 84 oz., was much enlarged and slightly hobnailed, texture firm.

The *spleen* weighed 24 oz.

The *pancreas* and *kidneys* were firm.

Explanation of the Plate.

A. Pseudo-ducts.
B. Coarse fibrosis.
C. Fine intercellular fibrosis.
D. Liver-cells.
Fixative, Perchloride of mercury.
Stain, Logwood, fuchsin, and orange *g*.

gall stones. Microscopically the fibrosis was seen to be fine, with some coarse strands in which pseudo-ducts were conspicuous.

These two cases are especially interesting because they correspond in most respects with results obtained experimentally by the ligature of the common duct in animals. They differ from Hanot's form chiefly in the existence of ascites and the occlusion of the duct. Cases of cirrhosis associated with gall stones have been recorded by Dr. S. Mackenzie,[1] Mr. D'Arcy Power,[2] and others.

The fact remains, however, that in the majority of cases of obstructive jaundice, even when prolonged, no cirrhosis follows.

The third case was that of a man of 42 (E. S., 1898), an indoor servant and heavy drinker, whose face bore testimony to his habits, who was admitted with deep jaundice of five weeks' duration, delirium, and pyrexia, and died the next day. There was no ascites. Post mortem the liver was found to be greatly enlarged, weighing 114 oz., smooth, bile-stained, free from perihepatitis, and the structure showing a fine fibrosis of imperfectly formed nuclear fibrous tissue. Many of the hepatic cells were destroyed, the others fatty. The kidneys were slightly granular and fatty. Here, again, the disease was not chronic, nor the jaundice of long standing; and the cause was alcoholic.

Although in view of the detailed description of such cases occurring in the experience of others it must be allowed that, clinically at all events, cirrhosis of the precise character of so-called biliary cirrhosis does arise from time to time, it is certain that it is rare, at least in this country. It is moreover still doubtful whether these rare examples are entirely distinct from hypertrophic cirrhosis of alcoholic origin, or merely an exceptional variety of it. At all events, the representative fibrosis of the liver with enlargement is in the vast majority of cases a hypertrophic cirrhosis set up by alcohol equally with the atrophic form; in other instances by syphilis, and not the rare condition described by the French pathologists.

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[1] *Trans. Path. Soc.*, 1889.

[2] *Ibid.* 1890.

SYPHILITIC CIRRHOSIS

The points at issue with regard to syphilitic cirrhosis are comparatively few.

The ordinary form which results from acquired syphilis in the tertiary stage has usually well marked characteristics when the liver is examined after death, although its identification during life is often extremely difficult, and mistakes in diagnosis are frequent with regard to it. In a typical instance the gummata in various stages, the massive wedges penetrating the liver substance at various points, the scarring and puckering and lobulation, the extensive perihepatitis, are striking and unmistakable morbid appearances.

Microscopically may be seen the gummatous cellular growth developing into fibrous tissue, or undergoing caseation. In the specimens represented here, for which I am indebted to my friend Dr.

Poynton, there is seen also well-marked endarteritis obliterans—a condition well known as the result of tertiary syphilis elsewhere, but, as far as I am aware, not observed directly in connection with hepatic syphilis, until it was discovered in this case by Mr. Plimmer, our able pathologist at St. Mary's.

In a paper by Hanot and Chauffard[1] this condition of arteritis is described as met with in hypertrophic cirrhosis with pigmentation and diabetes, to which latter the cirrhosis was regarded as secondary. The appearances found are shown in drawings which are appended to the paper.

In one case, however, scarring is mentioned, which is very suggestive of syphilitic affection.

In the second case it is said there was no history of syphilis, and the surface of the liver flat and finely granular.

One point which is not sufficiently recognised, and which I would emphasise, is that the morbid changes in syphilitic cirrhosis may exist in every degree Occasionally they are so slight as scarcely to alter the normal appearance of the organ; a few small scars, some thickening of the hepatic capsule, or slight perihepatitis may be the only morbid features perceptible externally on post-mortem examination.

This fact is of great importance in its bearing upon diagnosis. There may be no suspicious history, nothing whatever distinctive in the physical signs made

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[1] *Revue de Médecine*, 1882, 'Cirrhose hypertrophique pigmentaire dans le diabète.'

Plate IV.

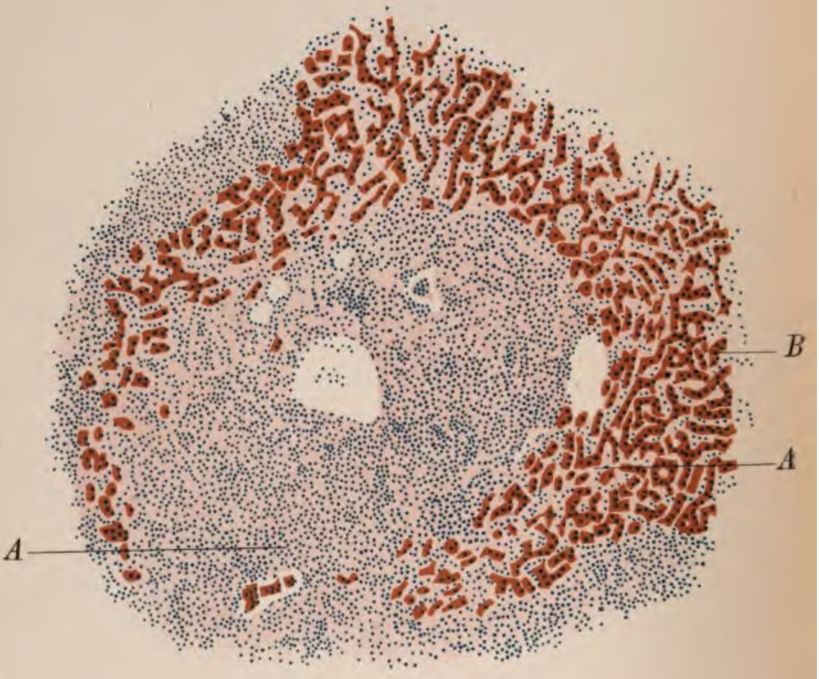

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PLATE IV

SYPHILITIC CIRRHOSIS OF THE LIVER—A GUMMA IN EARLY STAGE OF FORMATION

The patient, W. B., aged 37, was admitted for enlargement of the liver and spleen.

Later ascites developed, and paracentesis was frequently repeated.

The patient gradually lost strength and died, extremely emaciated.

Post-mortem.

The *liver* weighed 58 oz. There was extreme perihepatitis.
The *spleen* weighed 13 oz. The *kidneys* showed no cirrhosis.
The *lungs* contained numerous gummata.

Explanation of the Plate.

A. An area of small cell infiltration.
B. Cellular infiltration between the liver-cells.
Fixative, Perchloride of mercury.
Stain, Logwood, fuchsin, and orange *g*.

PLATE V

SYPHILITIC CIRRHOSIS OF THE LIVER—A GUMMA IN THE STAGE OF ORGANISATION

The patient, J. A., aged 56, was admitted for profound anæmia and hæmatemesis.

The liver was large and irregular. The spleen was large.

There was a syphilitic ulcer on the right forearm.

For nine months the patient was tapped about once a fortnight.

Death finally occurred after an attack of hæmatemesis and melæna.

Post-mortem.

The *liver* weighed 60 oz., showed great cirrhosis, was deformed, and contained organised gummata.

The *spleen* weighed 42½ oz.

The *lungs* contained gummata. The *pancreas* and *kidneys* were firm.

There was much perihepatitis and chronic peritonitis.

Explanation of the Plate.

A. Vascular fibrous tissue.

B. Liver substance.

Fixative, Perchloride of mercury.

Stain, Logwood, fuchsin, and orange *g*.

Plate V.

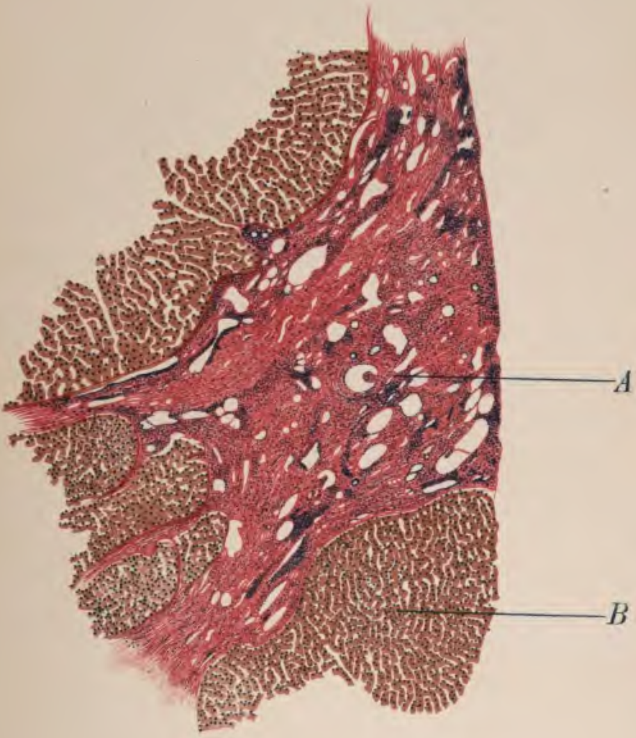

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Plate VI.

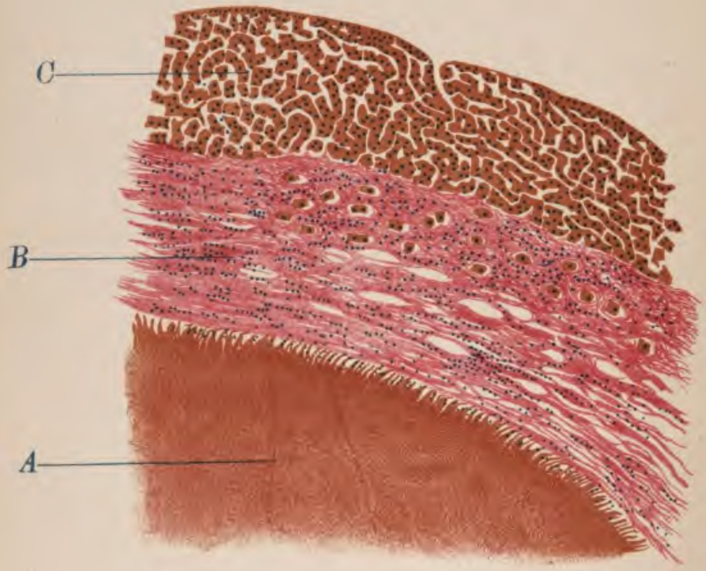

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PLATE VI

SYPHILITIC CIRRHOSIS OF THE LIVER IN AN ALCOHOLIC SUBJECT—GUMMATOUS DEGENERATED MATERIAL

The patient, A. S., a man aged 69, was admitted for ascites.

The liver was much enlarged, and there was slight jaundice.

He was tapped once. Facial erysipelas developed a week before death, which occurred two months after paracentesis.

Post-mortem.

Liver weight 74 oz., hobnailed. Showed great cirrhosis and numerous gummata.

Spleen weight 10 oz. *Pancreas* firm. *Kidneys* some fibrosis. *Cardiac muscle* pale and friable.

Explanation of Plate.

A. Gummatous material.

B. Fibrous tissue, containing some scattered liver-cells.

C. Liver tissue.

Fixative, Perchloride of mercury.

Stain, Logwood, fuchsin, and orange *g*.

PLATE VII

SYPHILIS OF THE LIVER—ENDARTERITIS OBLITERANS OF BRANCHES OF THE HEPATIC ARTERY

The patient, aged 22, died in uræmic convulsions.

Post-mortem.

Extensive endarteritis obliterans of the arteries in the kidneys, liver, and heart was found.

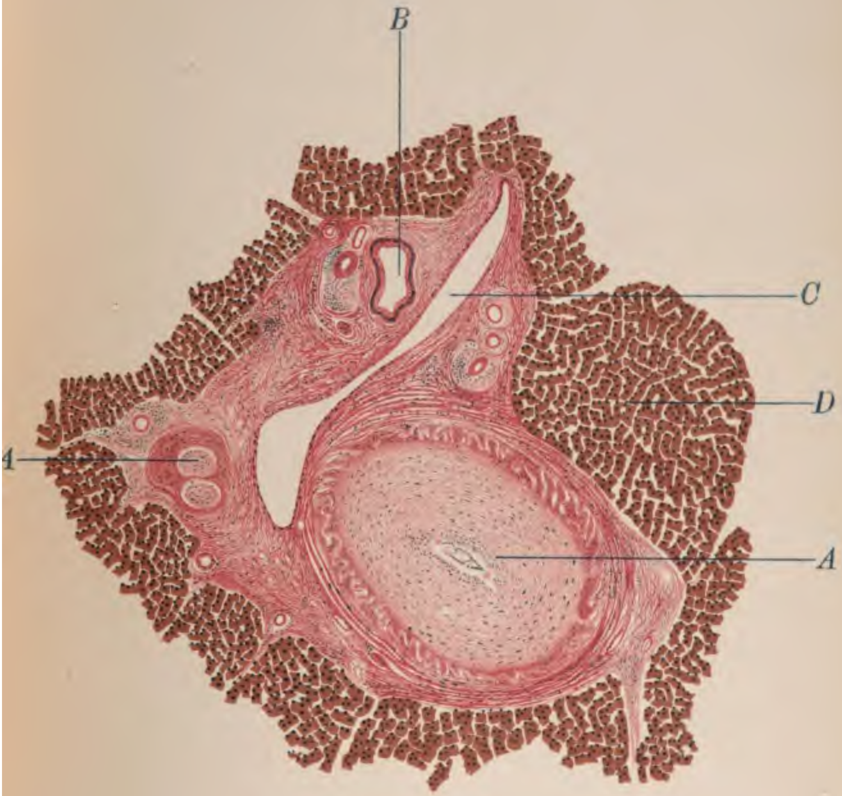
The *kidneys* contained numerous anæmic infarcts, the results of arterial occlusion.

Explanation of the Plate.

A Section across a Portal Canal.

A. Endarteritis in various stages.
B. A bile-duct.
C. A branch of the portal vein.
D. A liver tubule.
Fixative, Perchloride of mercury.
Stain, Logwood, fuchsin, and orange *g*.

Plate VII.



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out during life. The condition of the liver as ascertained by examination of the abdomen may correspond exactly with that of an ordinary alcoholic cirrhosis.

Another feature of syphilitic cirrhosis is the degree of perihepatitis and local peritonitis in the region of the liver.

This condition of general perihepatitis, with extension to neighbouring structures, is far more marked in connection with syphilitic disease than in any other condition.

The four most notable examples I have met with as I stated previously have been in this relation :—C. H.: Liver weight 134 oz.—J. R.: Liver weight 60 oz.—J. C.: Liver weight 88 oz.—W. B.: Liver weight 58 oz. In these the kidneys were normal except in one instance, when there was parenchymatous nephritis.

In the most extreme of all (W. B., 1899) the liver was studded with gummata in various stages, the capsule being covered with thick coating and adherent to the diaphragm, the abdominal wall, and the transverse colon.

The stomach and spleen were bound down by adhesions, the liver and viscera glued together in an inseparable mass. The liver was clearly the centre from which all neighbouring peritonitis spread. The kidneys were absolutely healthy. Yet there was no history of syphilis. The man was married and had healthy children.

This point of the great development of perihepatitis and local peritonitis, in many cases of syphilitic cirrhosis, has been imperfectly recognised Perihepatitis has been usually regarded as more especially associated with other forms.

Murchison, however, observed that it was met with in marked degree in syphilitic cirrhosis, and in connection with long-standing disease of the heart. He described it as accompanied by fibrous bands extending from the capsule into the liver structure and causing atrophy.

The fact of the association of extensive perihepatitis and local peritonitis with syphilitic cirrhosis is of considerable clinical importance.

There may be, as I have observed, no history of syphilis in these cases.

In ten of which I have notes—eight of which were fatal and the gummata found post mortem—in two only was there any definite history of syphilis; in two others syphilitic scars were found during life; but distinctive physical signs in the shape of irregularities or bossing of the surface were found in three only.

The usual condition disclosed by physical examination of the abdomen during life was a large liver smooth and slightly irregular and hard, or sometimes simply that the liver was small and contracted.

The syphilitic liver, then, is liable to be mistaken clinically, and as a matter of fact is, I believe, frequently mistaken during life, for the alcoholic liver; an error which in view of the remedial power of antisyphilitic remedies may be a disastrous one.

Let me repeat that one helpful symptom in such cases is the existence of signs of perihepatitis, pain, and tenderness over the liver, and pyrexia. When these exist in marked degree, and are at all persistent, they afford a presumption in favour of the syphilitic nature of the affection. The rise of temperature as shown in the charts on previous pages affords a valuable indication of the existence of perihepatitis.

Perihepatitis does indeed occur in pure alcoholic cases—and in connection with Bright's disease, but, according to my observation, it is not so constant or so extreme as in syphilitic cirrhosis.

Another undetermined question is as to the specific cirrhosis which arises in young children as a result of congenital syphilis. I have not met with any instance of gummatous development such as is found in the acquired disease, although the condition has been described by Ziegler. The smooth liver greatly enlarged offers no points of distinction from a liver enlarged from many other causes, and the diagnosis must depend upon the history or the existence of other signs of the disease.

Post mortem the microscope shows a distinct cellular invasion and early fibrosis spreading through the lobules between the cells in a diffuse and characteristic manner; there may be no thickening of capsule or marked external change perceptible.

In eight cases of cirrhosis in children between five and nine years of age of which I have careful records there was a reliable history of congenital syphilis in two only. In two there was one of alcoholism, and in the remaining four no cause could be made out. In connection with this subject of cirrhosis in children there are many interesting points, such as the effect of congenital obliteration or stenosis of the bile-ducts, as recorded by Dr. John Thomson,[1] the question of hereditary tendency as noted by Dr. Howard [2] and Dr. Finlayson,[3] and the connection of this with nervous lesions as noted by some observers. I think it is probable that congenital syphilis will furnish the key to the relation of these several conditions. With these matters, however, it is beside my present purpose to deal.

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[1] *Congenital Obliteration of Bile-ducts*, 1892.

[2] *Int. Journal Med. Soc.* 1887.

[3] *Glasgow Hospital Reports*, 1899.

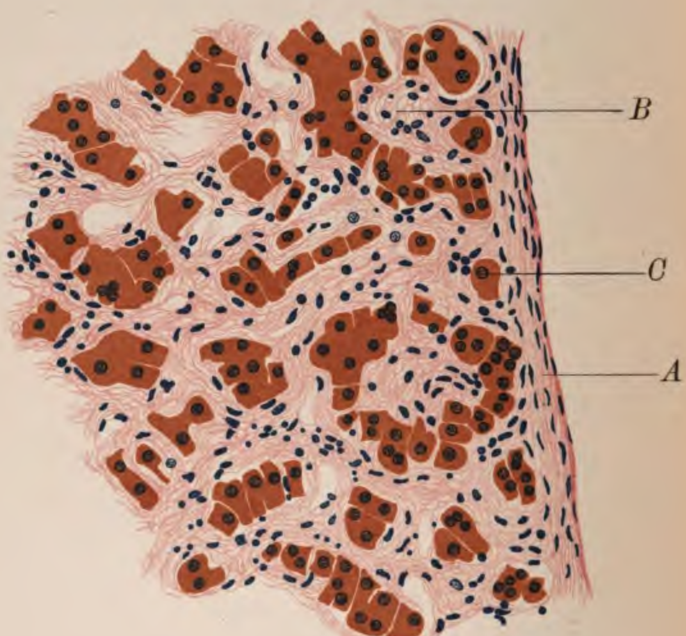

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PLATE VII (a)

SYPHILITIC CIRRHOSIS OF THE LIVER—CONGENITAL SYPHILIS SHOWING A DIFFUSE CELLULAR INFILTRATION AND EARLY FIBROSIS OF THE LIVER

From an infant of three weeks, who died of intense jaundice. The mother had lost all her children: some were premature. A previous child had also died of jaundice.

Post-mortem.

No obstruction of the hepatic ducts or common bile-duct.

The liver looked natural. The gall bladder contained blood and mucus.

Explanation of Plate.

A. The capsule of the liver not thickened.
B. The fine intercellular fibrosis.
C. The liver tissue.
Fixative, Perchloride of mercury.
Stain, Logwood, &c.

LECTURE II

General considerations with regard to the various forms of cirrhosis—Degeneration of heart muscle—Nature of the morbid change—Concurrent interstitial fibrosis of other organs — Kidneys — Spleen—Pancreas—Alcoholic cirrhosis only one phase of chronic alcoholism —Liability to erysipelas—To tuberculosis—Lines of demarcation between different cirrhoses too absolute—Sometimes more than one factor concerned—The fallacy of negative classification—Prognosis—Causes of too unfavourable view—Latent cirrhosis—Prognosis usually based on the advent of ascites—The standard of viability—Examples of favourable course in atrophic cirrhosis—Prognosis more hopeful in the hypertrophic form—Elements of favourable prognosis—Course of the successful cases.

THERE are certain further points of interest connected with the pathology of the different forms of cirrhosis discussed in the last lecture which deserve a passing consideration.

DEGENERATION OF HEART MUSCLE

The first of these, and perhaps the most important, is the association of marked degeneration of heart tissue with cirrhosis.

Degeneration of cardiac muscle and cardiac failure are, of course, acknowledged results of chronic alcoholism, and their importance in that relation has been stated by Dr. Graham Steele,[1] but the frequency of such degeneration and failure in connection with cirrhosis, its pathological significance, and its bearing upon prognosis, have, I think, been insufficiently appreciated.

Of the fifty-three fatal cases mentioned, the condition of the heart was especially noted in thirty-six.

In six of these fatty degeneration—in some instances extreme—was verified by microscopic examination; active myocarditis was observed in one; and in eight others the muscle was found pale, soft, and presumably fatty. In nine more there was atheroma of one or other of the cardiac valves.

There was therefore marked degeneration of heart muscle in seven, and in less degree in eight more—or fifteen in all—out of thirty-six, without counting the cases (nine more) in which atheroma of the valves only was recorded.

Of the fifteen cases of fatty degeneration, the liver was enlarged in ten instances, in five it was small and contracted. So that the tendency to degeneration is not limited either to the large or the small liver. In twelve the cirrhosis was distinctly traced to alcohol.

The frequent occurrence of these changes was first brought to my notice by my friend Dr. Poynton, and I am indebted to him for the following account of the condition of the heart muscle, and for the microscopic preparations from which the sketches in illustration have been taken.

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[1] *Med. Chronicle*, vol. xviii. 1893.

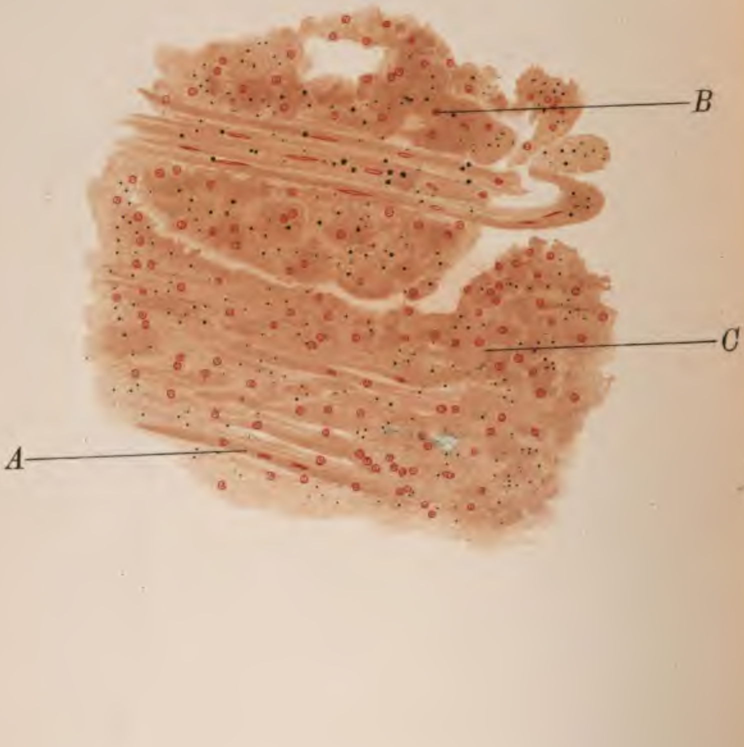
'The descriptions and figures are based upon an examination of six fatal cases of cirrhosis, all traced to alcoholism. In three the liver was enlarged, and in three contracted. The naked-eye appearance was one of undue softness and pallor, and the texture was abnormally friable. The changes were similar in all, but most extreme in one in which there was also peripheral neuritis. Microscopically these were seen to affect the muscular fibres rather than the interstitial tissues.

'Numerous dark granules ("brown atrophy") were always found in the muscle fibres, especially spreading from the poles of the nuclei. In addition to this, in several cases, the nuclei had undergone this dark granular change wholly or in part. Striation was often lost, and the fibres had a "ground glass" appearance. In other cases the striation was more evident than natural, giving a wirework appearance to the fibres. There was fragmentation of many fibres; fatty change was present in many cases, the fat globules being as a rule minute and finely scattered.

'The fibres were irregular in shape and size, and in the worst cases had lost all contour.

'The chief interstitial change was a fatty infiltration separating the fibres, and in some instances this condition was well marked. That these morbid changes were not dependent upon failure of general health as a result of the cirrhosis seems clear from the

Plate VIII.



Bale and Danielsson, Ltd., Del. and Lith.

PLATE VIII

A SECTION OF THE WALL OF THE LEFT VENTRICLE FROM A CASE OF ALCOHOLIC CIRRHOSIS OF THE LIVER

The patient, M. D., a woman aged 38, was suffering from atrophic cirrhosis, together with peripheral neuritis.

There was ascites, and she was tapped three times.

The pulse was feeble and the heart-sounds weak.

Death occurred after a brief period of delirium and coma.

Post-mortem.

The heart weighed 9 oz., and the muscle was extremely friable.

Explanation of the Plate.

A. Cardiac muscle fibres which still preserve their contour but have lost striation.

B. Nuclei of muscle fibres, the protoplasm of which has degenerated. Many of these nuclei are circular in outline.

C. Degenerated muscle protoplasm.

Fixative, Herrmann's fluid.

Stain, Safranin.

PLATE IX

CARDIAC MUSCLE FIBRES, FROM A CASE OF ALCOHOLIC CIRRHOSIS OF THE LIVER

The patient, S. R., a man aged 45, was a confirmed drunkard. He was admitted for ascites, and died suddenly.

Post-mortem.

The heart weighed 12 oz. The muscle was soft, dark, and flabby.

Explanation of Plate.

1. Transverse section of muscle fibres, showing 'brown atrophy.'

A. Nuclei.
B. Granules.
Stained with safranin.

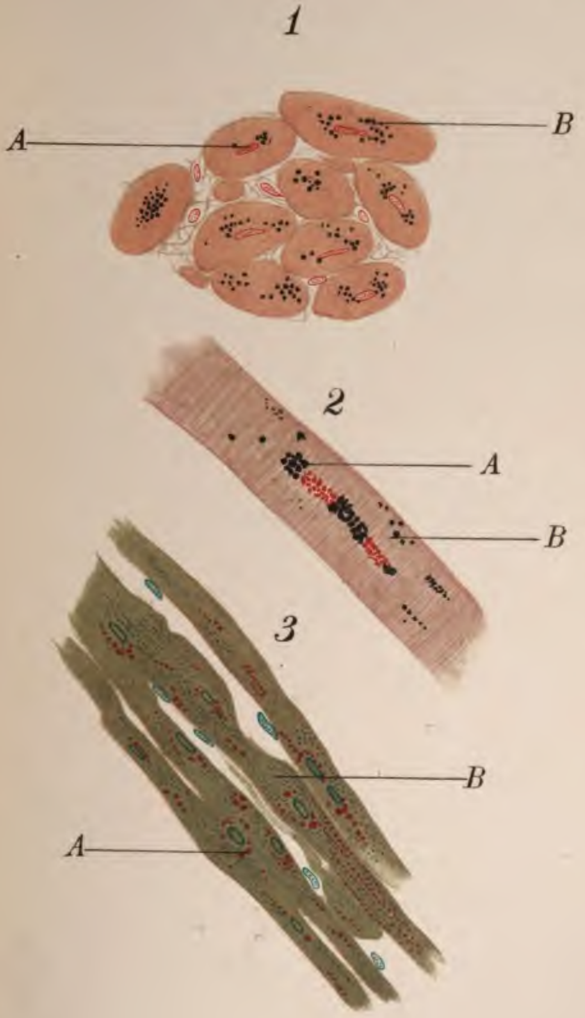
2. A single muscle fibre very highly magnified (obj. $\frac{1}{12}$; oc. 12), showing granular change in the nucleus as well as in the protoplasm.

A. Granules in the nucleus.
B. Granules in the protoplasm.
Stained with safranin.

3. Longitudinal section of muscle fibres, showing loss of striation and irregularity of contour.

Stained with methyl green.
A. Brown granules.
B. Fatty change.
Fixative, Herrmann's fluid.

Plate IX.



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fact that a similar appearance was found in the cardiac muscle of a drunkard who died of pneumonia and delirium tremens, in whom the liver was not cirrhotic.'

The frequent occurrence of serious degeneration of the cardiac muscle accounts for the fact that a certain proportion of these cases end suddenly in death by syncope; many more by rapid, if not sudden, heart failure.

In the fifty-three fatal cases mentioned the patient died suddenly from syncope (not the result of hæmorrhage) in six instances.

As I shall endeavour to show later, it is probable that the enfeebled cardiac condition may sometimes be the factor which determines the advent of ascites in alcoholic cirrhosis, and also be concerned in the development of the fibrotic change, and influence its character.

CONCURRENT INTERSTITIAL FIBROSIS OF OTHER ORGANS

Another important feature of the morbid state in cases of cirrhosis, or, at all events, of alcoholic cirrhosis, is that the interstitial fibrosis is not confined to the liver, although its most extreme development may be found there, and largely rule the pathological situation. It affects other organs, usually in like manner.

The kidneys suffer next to the liver in order. They were found to be distinctly affected in twenty-six of the fifty-three cases.

In five instances the kidneys were granular; 'moderately cirrhosed' or 'fibrosed' in thirteen; in five they are described as 'fibrosed' simply; in three there was parenchymatous nephritis.

Again, in twelve of these twenty-six cases in which the liver was markedly atrophic and small there was fibrotic change in the kidney in every instance, but in five only was the interstitial change advanced. In the remaining seven it was extremely slight.

The *spleen* was enlarged in twenty-three of these cases, eleven of these being in atrophic, twelve in hypertrophic cases, and its condition was noted as fibrosed in six.

The *pancreas* has been systematically examined in a few of the most recent cases only, viz. in fourteen.

It was distinctly cirrhosed in three instances, extremely hard or firmer than natural in ten; in the remaining case, which was one in which neither alcohol nor syphilis could be traced, the organ was large and soft.

In seven of the fourteen cases alcoholism was established; in two syphilis; in the remaining five the cause was not stated.

This association of cirrhosis of the pancreas with cirrhosis of the liver is of interest in connection with Hanot's[1] observation that hypertrophic cirrhosis was an occasional complication of diabetes, and also on account of the association of fibrosis and atrophy of the pancreas with diabetes independently.

In the cases here published the fibrosis of the pancreas was not limited to those cirrhoses of the liver in which that organ was enlarged, although these were in the majority, viz. nine, as compared with five of the atrophic form.

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[1] Hanot and Chauffard, *Revue de Médecine*, 1882.

In no case did diabetes occur, nor was sugar observed to appear in the urine ; but it is obvious that such a condition of pancreatic degeneration must seriously affect nutrition, and be a factor in the wasting which attends hepatic cirrhosis.

Alcoholic cirrhosis is indeed only one phase of chronic alcoholism ; yet we are apt to look upon it as if it were the single and only result ; as if all the evil effect of the poison were concentrated upon the liver alone. The heart, the kidneys, the spleen, the pancreas, the blood-vessels, the nervous system, all suffer, but usually in less degree. The great stress most commonly falls upon the organ which receives the poison first in full flood ; but the implication of the others constantly influences the course of the liver affection, upon which attention is mainly directed.

LIABILITY TO ERYSIPELAS

These records also show that persons suffering from cirrhosis, vulnerable in so many respects, suffer from a special liability to erysipelas, apparently beyond that common to all chronic exhaustive diseases.

Dr. Dreschfeld observed that erysipelas was not uncommon in Hanot's disease, but there is no exclusive association with biliary cirrhosis, or indeed with hypertrophic cirrhosis, as compared with the atrophic kind. In the fifty-three fatal cases of the last ten years death occurred from facial erysipelas in three. In two other instances of a previous period the fatal issue was due to the same cause, or five cases in all. In these five fatal cases of erysipelas complicating cirrhosis, the liver was enlarged in three—two of which were syphilitic, one alcoholic. In the remaining two the liver was contracted and hobnailed, and the affection of alcoholic origin.

LIABILITY TO TUBERCULOSIS

The close association of tuberculosis with cirrhosis of the liver is well known and acknowledged, and it is unnecessary for me to do more than allude to it here. It marks again the vulnerability of these subjects of degenerated tissues, and is due chiefly, I imagine, to the proved influence of alcohol in favouring tuberculous infection.[1]

DISTINCTIONS BETWEEN DIFFERENT FORMS TOO ABSOLUTE

The evidence which I adduced in the First Lecture supports, I think, the opinion which I ventured to express at the outset, viz. that the lines of demarcation between some of the different forms have been too rigidly drawn. They hold good with regard to typical examples only. Take, for instance, the mere question of size and weight. No strict line can be drawn between hypertrophic and atrophic forms—at any rate so far as alcoholic cirrhosis is concerned. The conclusion arrived at by Dr. Foxwell, that all sizes, from 100 oz. to 30 oz., are met with, is more than borne out by my series of cases, in which all sizes from 152 oz. to 18 oz. are found. And thus with regard to other symptoms. There are many cases in which the symptoms agree in some respects with those attributed to one form, in other respects with those attributed to another form. It is convenient to divide people morally into the two classes of sheep and goats; but, as a matter of fact, as I think we should all acknowledge, the majority of persons are neither the one nor the other, but something of each. There are many hybrids. And so it is with regard to our conceptions of disease. We look for a strict separation into classes, and the expectation of close conformity to given types is apt to warp our intelligence.

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[1] Dickinson, *Med. Chir. Trans.* vol. lvi. 1873.

It is thus with reference to cirrhoses: they are met with in typical forms, but occur also in various degrees of modification between them.

We assume one factor to be alone engaged, and expect each case to correspond to the authorised description. Yet the acknowledged causes—alcohol, syphilis, malaria, cardiac disease—are just those which, although they may usually act alone and produce simple results, are, from the very nature of things, liable to be associated; to act together in varying degree upon the same subjects, and produce results more or less complex. In these instances the effect of the relative influences at work is not easily estimated. And then there is the fallacy of negative classification. If no alcoholic history is traced, the case is called *non-alcoholic.* If no history of syphilis, *non-syphilitic.* The fallacy of this with regard to syphilis I have shown; and that it exists largely with regard to alcohol we all know well.

Occasionally—as in certain cases of cirrhosis in children—no adequate cause can be discovered. And there are others, again, which differ from all recognised forms, and from any combination of the characters of the acknowledged varieties.

Thus in a case recently under the care of my colleague Dr. Phillips, a young man of thirty-seven (G. M.)—with enlarged liver and extreme ascites, but no jaundice—the history appeared absolutely to exclude alcoholism and syphilis. He died at the end of a year of hæmatemesis, having been tapped thirty-five times.

Post-mortem examination disclosed a large hobnailed liver, with small patches of perihepatitis. Microscopically multilobular, and fine monolobular, fibrosis, with active cell proliferation; no gross portal obstruction, no sign of syphilis, or of general alcoholic degeneration; some fibrosis of the kidney, but this not extreme; the heart muscle firm and healthy. The source of cirrhosis remained undetermined.

PROGNOSIS

The prognosis in cirrhosis varies according to the particular form in which it appears, and the conditions associated with it. Yet, as set forth by authority in books on medicine, it is almost universally, and absolutely, unfavourable. Its fatality is regarded as inevitable, and the end speedy.

Thierfelder, speaking of cirrhosis generally, says: 'In the cases which have been accurately diagnosticated the termination is without exception *death*.' Frerichs, Niemeyer, Murchison, spoke in like fashion; and more recent authorities are almost equally pessimistic.

This unfavourable verdict, more positive than the actual facts seem to me to warrant, has, I think, arisen partly from the use of the generic term *cirrhosis*, when the condition really intended to be indicated is one form of the disease only, viz. *the atrophic contracted liver of Laennec*. Thus, the occurrence of ascites, so significant of evil in that condition, has been assumed to be equally serious in hypertrophic or syphilitic cirrhosis where its import is less grave.

The morbid conditions met with in cirrhosis are to a large extent incurable. The congestion may be relieved, the perihepatitis subdued, gummatous change be arrested, ascitic pressure removed; but fibrous tissue is a permanent formation; it is, however, in itself harmless; it injures only by pressure; and it may exist within certain limits for long without fatal issue. In some degrees of it, the glandular structure of the liver is not so extensively injured as to render life impossible, and if the obstruction to the direct portal flow is eased by compensatory channels, fair health may be maintained for a season.

Cirrhosis is not a disease of rapid development, but of slow and gradual progress. It may advance silently and steadily for years before its presence is discovered. The liver is frequently found indurated to an extreme degree, and usually enlarged, in persons who have died from other causes, without the patient having been known to suffer from any marked symptoms of such affection during life. Dr. Fagge[1] noted that the age of such persons, thus found accidentally to have been suffering from cirrhosis without discovery, was on an average five years in excess of the average age of those who died directly of the disease. This would seem to show that cirrhosis is often quiescent before reaching its final stage. The elaborate statistics of Dr. Rolleston and Dr. Fenton[2] give, however, somewhat different results, and make the patients with latent cirrhosis slightly younger than those who die of the disease.

Further, as the existence of cirrhosis is commonly betrayed by the supervention of ascites, the estimation of its future course is ordinarily made towards the extreme end of it. If the disease could be recognised earlier the prognosis would be in corresponding degree less immediately unfavourable.

I have now under my care a man of upwards of fifty, an inveterate spirit drinker, whom I have known for twenty years, who has a greatly enlarged liver—reaching four fingers' breadths below the ensiform cartilage—and as hard as wood. He came under my care for influenza; and although I knew his habits of life, I knew nothing of any hepatic disorder. He has no ascites—no special symptoms of cirrhosis, beyond a passing jaundice, some loss of flesh, varicose stigmata on the face—and the large hard liver.

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[1] *Principles and Practice of Medicine*, 3rd ed. vol. ii. p. 370.

[2] *Birmingham Medical Review*, October 1896.

In another instance, an old country gentleman of my acquaintance, of alcoholic habits, had, to my knowledge, a large indurated liver for many years. He enjoyed fair health up to seventy, and then died of hæmatemesis, supervening upon an attack of pneumonia. He never had ascites or any special signs of cirrhosis, except the enlarged hard liver and a certain alcoholic aspect.

As I write this I have under observation a young man of twenty-nine, of extreme alcoholic habits, taking a bottle of brandy a day, who has already a hard, enlarged liver, extending two inches below the edge of the ribs. He has no other symptoms except some dyspepsia and palpitation, and a slightly dilated heart. There can, I presume, be no doubt that he has early cirrhosis. He is fat and well nourished, and the prognosis depends upon the future regimen.

The prognosis in cirrhosis is, however, almost invariably made with regard to the advent of ascites, and turns upon this symptom, and is, indeed, usually entirely based upon it. The observation of Dr. Bristowe in 1892,[1] that most medical men of that time regarded the development of ascites in connection with liver disease as of fatal omen—or at any rate as the beginning of the end—still, I believe, holds true. Yet some masters in medicine have expressed more hopeful views.

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[1] *Brit. Med. Journal*, April 23, 1892.

Dr. Frederick Roberts[1] and Dr. Austin Flint[2] have shown that the supervention of ascites is not necessarily a sentence of immediate death, and each adduced a series of cases in proof of it.

It is not disputed, I think, that, occasionally, the course of cirrhosis—even after ascites has supervened—is in some cases prolonged for months, and in a few rare instances for a certain number of years.

Hitherto, however, as far as I know, no satisfactory discrimination of the conditions in which a favourable issue may be regarded as possible has been established.

The first point to lay down is that when the cirrhosis is of the atrophic contracted kind, traceable to alcoholic excess, the prognosis is the least favourable. If ascites has supervened, as is usually the case when the diagnosis is made, it probably indicates that the disease has reached its final stage. Much, however, depends upon the condition of general nutrition. In proportion as this is satisfactory, the prognosis is more hopeful of respite. If the emaciation and cachexia are advanced, the condition is one of proximate gravity.

The course of some of the more favourable cases of atrophic cirrhosis which survive for a time is exemplified by that of a woman previously mentioned who died of intercurrent bronchitis before the liver was so extensively injured as to seriously impair general nutrition.

She was a small, tolerably plump, slightly anæmic woman of fifty-three (S. D., 1891), admitted for ascites and hæmatemesis. She had long been a steady drinker, with her husband as boon companion. He was a carpenter employed in the Medical School, whose alcoholic excesses became well known to me when I was brought into contact with him there, during my term of office as Dean. The woman admitted that she took half a pint of gin every day, in addition to stout, and had done so for the last thirty years. She was tapped twice, about 600 oz. being drawn off. The fluid did not return, her general condition rapidly improved, and she went out at the end of two months in fair health and nutrition.

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[1] *Practitioner*, 1872.

[2] *Brit. Med. Journal*, 1883.

Nine months later she was readmitted. The ascites had returned, and the hard edge of the liver could be felt two inches below the costal margin. She was tapped ten days after admission and 198 oz. withdrawn. There was no return of the effusion, but a sharp attack of acute pleuro-pneumonia developed —probably influenzal—with extensive consolidation, pleuritic rub, rusty sputum, high temperature, and delirium. Her case was judged to be hopeless; but under the free use of digitalis in doses of fifteen drops every four hours, given by my earnest house physician at the time, and a temporary dispensation of an allowance of wine, a crisis came and, to our extreme surprise, she recovered. A month later she went out of hospital for the second time in a state of comparative health.

A month later still she was admitted for the third time—on this occasion with an acute bronchitis of great severity, contracted by exposure in the extremely bitter weather which prevailed at the time.

The post-mortem examination disclosed a contracted, typically hobnailed, cirrhotic liver, weighing 38 oz., with thickened capsule, but no general perihepatitis or peritonitis, no thickening of, or adhesions to, neighbouring structures. It was calculated from general appearances, aided by microscopic examination, that about one-half of the liver tissue was efficient, the cells being normal and active.

The kidneys showed commencing granulation, but were not much contracted, weighing 4½ oz. and 5¼ oz. respectively. The heart showed advanced fatty change, with so-called tabby-cat striation in a marked degree.

This case is of importance, since the patient was not in a condition of emaciation, and regained fair general health. The fact that she recovered from a severe attack of pleuro-pneumonia showed that the liver damage was not so extreme as to be incompatible with a state of nutrition and vigour equal to the strain of acute intercurrent disease and restoration to the previous degree of health afterwards.

The following is another example of the more favourable course in atrophic cirrhosis:

A. D., æt. forty-three, a cabman (formerly a potboy), of intemperate habits, was admitted to St. Mary's Hospital on April 4, 1894, suffering from ascites. The man was plump, soft of skin, flabby of flesh. The abdomen was much distended with fluid, measuring forty and a quarter inches in circumference. The hard edge of the liver could just be felt below the margin of the ribs, the heart-sounds gave a slight apex bruit, and the apex was pushed up to the fourth space. There was no hypertrophy. A few medium moist râles were audible at the base of the lungs. The urine had a specific gravity of 1037 and was free from albumen. He was tapped nineteen times between April and August, nearly 3,000 oz. being removed. On August 9 he was tapped for the last time. The edge of the liver was not to be felt. The fluid did not return, and he was discharged on September 20, apparently well, after being under treatment for six months. On June 18 of the following year he called to report himself. He complained of loss of appetite, but he still had a fair amount of subcutaneous fat, and was moderately well nourished; there was no return of ascites. The liver could be felt, just below the costal margin, extremely hard.

In the following August he was readmitted for hæmatemesis. The liver could just be felt. There was no ascites. The urine was of specific gravity 1020, with a trace of albumen, and there was a systolic bruit in the aortic area. Hæmatemesis recurred, and he died the following day—a year and a half after coming under treatment for ascites.

At the post-mortem examination the liver was found to be contracted, weighing 48½ oz., markedly hobnailed and tough, covered with thick false membrane, the spleen enlarged, the kidneys enlarged slightly, otherwise normal. The valves of the heart atheromatous, otherwise the heart was normal. There was no general peritonitis.

Again, a woman of thirty-nine (E. M.), an acknowledged spirit drinker, was admitted into St. Mary's for jaundice and ascites in July 1893; was tapped twice; and, the ascites not returning, she went out apparently well, to be readmitted in January 1894, six months later, with a return of ascites. She was again tapped, once only; and remained without much return of fluid until January 1895, when she was again admitted and tapped a fourth time.

By some mischance, which could not be traced, suppurative peritonitis supervened, and the patient died—one and a half year after first coming under treatment for ascites.

The post-mortem was most instructive, on account of the extremely small size of the liver, which weighed only 18 oz., instead of 50 oz., and was typically hobnailed, with abundant adhesions between the liver and neighbouring organs, great thickening and shortening of the large intestine—the ascending colon being only two inches in length—and the fact that no morbid changes were found in the kidneys.

Take, again, another case (that of a man who came under the care of my colleague, Sir W. Broadbent), E. P., a florid but fairly healthy-looking man aged thirty-three, with a history of alcoholism, but none of syphilis.

He was tapped eleven times between 1893 and 1896, a space of three years, coming into hospital eight times for the purpose. The liver was small, only just felt at first; and at the last visit, in June 1896, its size could not be made out. The man left hospital at that time apparently well, and was lost sight of afterwards.

Take, again, a case such as this:

The patient (Q. T., Sir W. B., December 1893), a man æt. thirty-five, was tapped eleven times for ascites which had been first noticed in July 1892. It being found impossible to remove all the fluid, a localised collection was suspected, and for this laparotomy was performed in December 1893, nearly a year and a half after the first appearance of ascites. The diagnosis of a localised collection was verified; chronic peritoneal adhesions were found shutting in fluid between the stomach and diaphragm. The operation, however, proved fatal.

Post-mortem: Advanced atrophic cirrhosis, perihepatitis, perisplenitis, and perienteritis were found. The kidneys were unaffected.

In this case perihepatitis may have played a part in the production of the abdominal dropsy. It seems to me immaterial, except as it might affect the possibility of a favourable issue, whether the fluid was poured out as the direct result of portal obstruction, or of an associated local peritonitis, due to the engorgement of the hepatic peritoneum from this same obstruction, or to alcoholic irritation. However this may have been, the case was one of advanced cirrhosis and ascites, and the kidneys were unaffected.

In my notes I find a number of other cases of atrophic cirrhosis in which life has been prolonged for considerable periods after the first appearance of ascites.

Apart, however, from these observations of mine, there is evidence from other sources as to the occasional prolongation of life in atrophic cirrhosis for a considerable period after the betrayal of its existence by the occurrence of dropsy.

One such is recorded by Dr. Bristowe,[1] in which the patient remained well for six years. The man was a drinker, had alcoholic neuritis, and the liver was not enlarged.

The most notable case I have found on record is one given by Dr. Hawkins in his important article on cirrhosis in 'Allbutt's System of Medicine.'

The patient, a free drinker, came under the care of Dr. Murchison for alcoholic cirrhosis and ascites. From that time he became a total abstainer and for twelve years enjoyed fair health. At the end of that period he came into St. Thomas's Hospital with granular kidney and peritonitis, of which he died. The post-mortem examination showed a hobnailed liver with multilobular fibrosis and thickened capsule adherent to the diaphragm.

In the hypertrophic form, however, the prognosis is less gloomy. In the majority of cases, indeed, the disease progresses to a fatal ending, but far less certainly, and, as I have seen it, less rapidly, than in the contracted form. As I pointed out in 1892,[1] it is the large liver which gives promise of recovery. Curiously enough, however, according to Dr. Rolleston and Dr. Fenton's statistics,[2] and those of Dr. Pitt,[3] patients with enlarged livers, who die, are younger than patients who die with contracted livers. There is, however, no incompatibility between the two statements. The patients with enlarged livers are younger than those with contracted liver; if they die they die younger probably.

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[1] *Brit. Med. Journ.* April 1892.

This does not, however, affect the question whether more of these recover, or prove that, although they die younger on the average, some do not run a protracted course.

Again the cirrhosis of the enlarged liver is said to be more acute, and this I believe is so, as I shall show later. But this refers only to its development; its after course may be prolonged.

In some instances, no doubt, the estimation of the size of the liver during life is liable to error. A contracted liver displaced downwards may be mistaken for actual enlargement, especially in the case of women, and when ascites is present; and, on the contrary, a large liver may be mistaken during life for a small contracted one, when it is bound by adhesions to the diaphragm and tucked away under it. Each of these cases has happened within my knowledge; but once only, I think. In all other cases which have ended fatally and the condition of the liver has been verified by post-mortem examination, the diagnosis of enlargement or contraction has proved to be correct. Be this how it may, however, to put it broadly and speaking clinically, the cases which do well are those in which the liver, apparently large, exposes a flat hard surface to the abdominal front, extending far below the edge of the ribs, and sometimes down to the umbilicus.

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[1] *Brit. Med. Journal*, November 10, 1892.

[2] *Birmingham Med. Rev.* October 1896.

[3] *Path. Trans.* vol. xi.

In three cases of recovery from alcoholic cirrhosis with ascites, recorded by Murchison, the liver was enlarged. In another attributed to mitral disease of the heart the liver was enlarged. In one of the four cases observed by Dr. Bristowe the liver was greatly enlarged. In two cases recorded by Dr. Dickinson,[1] in which recovery took place after extreme ascites developed, and in one of which the diagnosis was verified on death from cerebral abscess two years afterwards, the liver was described as hypertrophic.

In eleven other cases, which I have culled from various authorities, in which recovery is stated to have taken place, and the patient to have regained apparent health, for periods varying from one year to eighteen years, the liver was stated to be enlarged in six, contracted in one, and the condition not stated in four.

This fact of the more favourable prognosis in cirrhosis when the liver is enlarged was recognised by Hanot and Gilbert,[2] who, writing of alcoholic cirrhosis, affirmed that the hypertrophic form was the most curable, and that all the most successful cases they had seen were of this type.

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[1] *Allbutt's System of Medicine*, vol. v. p. 692.

[2] *Archiv. Général de Médecine*, 1890, vol. clxvi. p. 250.

In my address in 1892 I enumerated eight cases of hepatic ascites, in which relief of the ascites and restoration of general health took place.

To these cases of recovery, temporary or prolonged, I may add three more, making eleven in all.

These are exclusive of those instances in which there was mere relief of symptoms, sufficient to permit of discharge from hospital, and private cases which were not traced afterwards.

Of these eleven cases the liver was greatly enlarged in seven, considerably enlarged in one, slightly enlarged in two, and in one the condition of the liver was not ascertained.

If I add to these cases nineteen others, in which relief was such as to warrant discharge from hospital, or, in private cases, cessation of attendance, there are thirty in which recovery, either temporary or prolonged, resulted. I may remark with regard to the St. Mary's cases that they were not merely tapped and discharged at once, but kept in for considerable periods, until recovery appeared to be stable and maintained. Of these thirty the liver was markedly enlarged in nineteen instances, normal or slightly enlarged in three, smaller than normal in five, and in the remaining three the condition was not ascertained.

So that here, again, in nearly two-thirds of the cases in which good was effected by treatment, the liver was enlarged.

One salient feature of the cases which are capable of relief or recovery, then, is that the liver is enlarged.

The next question that offers itself is, whether any of these were cases of genuine alcoholic cirrhosis, or were they merely instances of syphilitic fibrosis?

A certain proportion of cases of cirrhosis with enlargement which recover are undoubtedly syphilitic, having a history or marks of syphilis, or yielding to antisyphilitic treatment; as, for example, a case recorded by Murchison, one by Sir Grainger Stewart, and three under my own care. Often, however, in syphilitic fibrosis contraction proceeds together with gummatous formation, and no great enlargement takes place.

It must, however, be borne in mind, as I have said before, that there is a close social relation between alcohol and syphilis, and that in some instances both these factors may be engaged.

Further, in certain instances, as I know by grievous mistakes in diagnosis, syphilis exists when there is no history of it to be obtained; where there is nothing in the physical character of the liver, as far as can be ascertained during life, to suggest it even. As I urged before, there may be no history of syphilitic infection, no irregularity of surface, no enlargement or marked contraction discovered, and yet the condition be syphilitic. There is, however, sometimes, as in the instance already mentioned, extensive persistent or recurrent perihepatitis, which, in the absence of distinct renal disease, is in itself suggestive of syphilitic origin; and its occurrence should always arouse suspicion. Apart from these cases, however, of syphilitic or mixed syphilitic and alcoholic hypertrophic cirrhosis, there are, as has been shown already, instances in which the disease is of purely alcoholic origin. These also are to be included amongst the cases of hypertrophic cirrhosis which now and again recover. Syphilitic cases, it is generally allowed, occasionally recover. A certain proportion of cases of hypertrophic cirrhosis, also, in which no cause except alcoholic stimulation can be traced—in which syphilitic remedies are useless, and even harmful—from time to time recover sufficiently to enjoy apparent health for years.

Three cases recorded by Murchison were alcoholic in origin. In six cases of hypertrophic cirrhosis under my care, which recovered for long periods, four were judged to be purely alcoholic, one alcoholic and probably syphilitic also, and one syphilitic and probably also alcoholic.

Another condition, in addition to the hypertrophic enlargement which marks the cases promising recovery, is the state of general nutrition. The patients who recover are, as a rule, comparatively healthy in appearance, not emaciated nor cachectic.

The significance of this lies of course in the evidence it affords of the efficiency of the vital functions of the liver. It shows that sufficient gland structure remains intact to serve the purpose of nutrition.

In some cases of syphilitic affection which get well the cachectic state may be marked. In the pure alcoholic cases, however, all the patients who have recovered within my personal knowledge have been in fair condition generally, not distinctly cachectic or greatly emaciated.

Another feature of the cases which are of more favourable prognosis is that they are as a rule younger than those which end fatally. Of the eleven cases of recovery which I adduced, I omit five in which the further history was imperfectly traced. In the six others the later career was ascertained for periods ranging from one to eleven and a half years. The average age is just over thirty-nine.

In thirty-one cases of recovery recorded by others the average age is, curiously enough, almost exactly the same, just under thirty-nine.

Further, in the enlarged liver we find a texture tough and hard, but not of that leathery hardness and toughness found in the contracted form; there is little tendency to contraction, unless it be some drawing in of the surface at interlobular points, giving the appearance of slight granulation.

Microscopically, the connective tissue development is as a rule in a more active stage than in the atrophic form, the fibrosis more diffuse and finer, and one in which cell proliferation is conspicuous. Thus in the case (S. No. 8) of extreme alcoholism, in which the liver weighed 114 oz., the liver was smooth, without thickening of the capsule, or any peritonitis; the connective tissue cellular elements greatly increased, but the fibrous tissue imperfectly formed. In another (G. M., Case 11), not traced to alcohol, in which the liver weighed 72 oz., the same active cell proliferation was observed.

In the case of the boy (A. A.) with enormously enlarged liver, previously described as probably a cirrhosis of congenital syphilis, this appearance of active cell proliferation and imperfectly developed fibrosis was most conspicuous. In a case of hypertrophic alcoholic cirrhosis recorded by Murchison, in which the liver weighed 80 oz., the nuclear element was observed to be extremely abundant.

This sign of an active process appears to prevail chiefly in hypertrophied forms, and suggests a more acute condition than the mature fibrosis of the contracted form, which bears evidence of a chronic and advanced development. Possibly in some instances of early atrophic cirrhosis nuclear growth may be conspicuous; in cases, however, which progress to a final stage, and in which death results directly from the liver affection, the contrast between the two conditions of fibrous change is generally distinctly marked.

Dr. Price,[1] as the result of his investigation of 143 cases of cirrhosis, came to the same conclusion, viz. that the large cirrhotic livers represent a more acute form of the disease than do the small.

If these conclusions are confirmed, I think that some of these cases of hypertrophic alcoholic cirrhosis, in younger subjects, might be termed acute cirrhosis; set up by more acute and extreme drinking, probably of more dilute alcohol in large quantity, as contrasted with the cirrhosis of older persons whose steady and protracted, but less intense, drinking, of more concentrated spirit spread over many years, produces a more dense and mature fibrosis, with greater destruction of secreting tissue.

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[1] *Guy's Hospital Reports*, Series III. vol. xxvii.

The characteristics of these cases of cirrhosis which are amenable to treatment, and run a more favourable course to arrest, or recovery—viz. an enlarged liver, the earlier age of the subjects, and the fact that they are generally in a condition of good nutrition, not emaciated, cachectic, alcoholic wrecks—are, I think, important. They all point to the conclusion that the morbid change is of a more active character than in the less favourable contracted form of cirrhosis, in which the invading tissue is of gradual and slower growth, and in a more advanced state of formation.

To sum up. The distinctive features of the favourable cases are:

1. The liver is large, the cirrhosis probably more acute.

2. The subjects are on the average younger than in the less favourable cases of contracted liver.

3. They are in a condition of fairly good nutrition.

Lastly, they are, as a rule, either exceptionally hard drinkers, acute drinkers, or they are syphilitic.

It may be asked with regard to these cases of apparent recovery in syphilitic and alcoholic cirrhosis, as shown by subsidence of ascites and recovery of general health, how far this remained permanent.

There is nothing more difficult, as every hospital physician knows, than to trace out cases to their conclusion. Leaving aside all which have been lost sight of quickly, I have been able to follow out the history with some completeness in six instances, viz. for considerable periods, varying from one to twelve years, two, to my knowledge, surviving to this day. Others I have observed for less, although for considerable periods, at the close of which the recovery remained unimpaired. To show more completely the course and character of these cases I shall state some of their salient features in the next lecture.

LECTURE III

Treatment—The chief objects to be sought—Maintenance of the *status quo*—Easing the work of the crippled machinery—Compensation for hepatic inefficiency—Relief of portal circulation—Diet—Saline laxatives—Gastric antiseptics—Tonics—Mercury and the iodides—Digitalis in cardiac debility and dropsy—Removal of ascites—Evil effects of abdominal distension on organs—Purgatives—Diuretics—Restriction of drink—Paracentesis—Former objections to it—Importance of its early use—Cases in illustration—Part played by the heart in hepatic ascites—Cardiac inefficiency a factor—Cases in illustration—Conclusion.

The treatment of cirrhosis resolves itself, chiefly, into an attempt to preserve the *status quo*, as far as the liver is concerned; to prevent the disease advancing; and to relieve any difficulties or imperfections which may arise in the bodily condition, as the result of the hepatic inefficiency.

No remedy will remove the fibrous tissue which has choked the liver-cells. No drug will restore the cells thus destroyed.

The only condition in which the invading agent can be removed, to some extent, by treatment, is in the early stage of syphilitic cirrhosis; when the gummata are not yet organised, and the arteritis and perihepatic inflammation are comparatively recent.

For the rest, all that can be done is to attempt to relieve the injured machinery, and supplement its powers, so that the crippled organism may survive. When the injury is not extreme this can be done, and the cirrhotic patient hold on to life, in fair health and comfort, sometimes for years.

With regard to the first great canon of treatment, the prevention of increase of fibrosis and destruction of liver tissue, absolute abstention from alcohol, and all stimulating ingesta, is of course the prime necessity. It is as essential in the syphilitic, malarial, and nephritic cases as in those of direct alcoholic origin. There is, however, one reservation to be made. When the last stage of emaciation and exhaustion has been reached, the total privation of alcohol, as we often see in hospital wards, hastens the final collapse.

And in the same way all foods which tax especially the physiological function of the liver, and therefore tend to embarrass and engorge it, such as those rich in fats and sugar, should also be given in reduced quantity, or altogether prohibited.

The diet should be restricted to milk, eggs, the simpler proteids, bread, and fresh fruit and vegetables.

It is to be remembered that not only is the passage of nutriment prepared by gastric and intestinal digestion, for distribution by the systemic vessels, interfered with by the portal obstruction, but that the glandular structure of the liver is partly destroyed, and its physiological function necessarily impaired also.

We are too apt, I think, to look upon the question of nutrition in cirrhosis as one of mere difficulty of transport; whereas it is largely one of preparation for absorption and assimilation, and of elimination. The secretion of bile; the glycogenic conversion of the carbonaceous elements of food; the curious part played by the liver in the metabolism of nitrogenous waste, are all interfered with; and the general nutrition of the body suffers in like degree.

In all cases, therefore, where signs of defective nutrition appear, such as wasting, anæmia, debility, it is well to supplement the work of the digestive machinery by artificial means. Predigested foods, especially pancreatised preparations, or pancreatin in keratin capsules, given with food, are useful aids to the defective liver function.

Further, taking into consideration the embarrassment of the portal circulation, and the consequent engorgement of all the viscera, whose venous drain lies through the portal, this condition should be relieved by mild laxatives, such as salts of soda and magnesia. Rhubarb, or cascara, or other unirritating drug of the kind, may be added to quicken the action of the salines, if necessary; but anything like hard purging should be avoided. To these laxatives iron also, or the bitter tonics, as quinine or strychnine, may be added on occasion.

In the flatulent dyspepsia which so often troubles the cirrhotic patient, full doses of bicarbonate of soda with aromatics, or hyposulphite of soda, or creosote, or carbolic acid, may be given to arrest fermentation.

There are, however, three drugs which, more than all others, are capable of signal service in appropriate cases: they are mercury, iodide of potassium, and digitalis.

Mercury and the iodides, especially the latter, sometimes effect a marvellous change in syphilitic cirrhosis, in virtue of their power in arresting gummatous formation, endarteritis, perihepatitis, and their consequences. The usual rule laid down is that, if there is any reason to suppose the affection to be syphilitic, iodide of potassium should be given. I would, however, urge that these remedies should be given, not only when there is reason to suppose the cause to be syphilitic, but given also steadily in all cases where, by any remote possibility, the syphilitic factor may be concerned; even when the patient is convicted of alcoholism. As I have pointed out, the social connection between syphilisation and alcoholism is naturally a close one; moreover, the syphilitic liver frequently closely imitates the alcoholic liver, and a mistake may be fatal.

In a case recently under my care the liver was studded with gummata, bound down by abundant perihepatitis, and tucked away under the diaphragm, so as to present all the physical signs of a small contracted liver. The patient was a butler: there was no history of syphilis to be made out on minute inquiry. He was a married man with a family of healthy children.

Antisyphilitics were given at first; and then, unfortunately, under the strong presumption of alcoholism, they were abandoned, and the chance of recovery lost.

Digitalis, again, is of material service in those numerous cases in which there is weakness and dilatation of the heart. The enfeebled, dilated state of the heart, so commonly present in cirrhosis, and the condition of the cardiac muscle found after death, explain the often beneficial influence of digitalis and like cardiac tonics.

THE TREATMENT OF ASCITES

The main interest in the treatment of cirrhosis centres in the ascites. In the majority of cases, it is this symptom which betrays the existence of the disease. It is a sign that tension in the portal vein, and its tributaries in the peritoneum, has been raised to the oozing point, or has set up inflammatory effusion ; that the flow of nutriment from the alimentary canal into the general circulation is gravely impeded ; that the liver function is seriously impaired. It is not, however, from this point of view alone that the advent of ascites becomes so important.

It is rather the mechanical effect of the distension of the abdomen with fluid which is so serious. The diaphragm is pushed upwards, so that abdominal respiration almost ceases, and the lungs are imperfectly inflated. Congestion, collapse, and basal bronchitis follow, and not infrequently effusion into the pleura in addition.

The heart is pressed upwards and embarrassed, and the impediment to the circulation, already existing in the pulmonary and systemic vessels, thereby greatly increased. The movements of the stomach are hampered, and the circulation in that chief organ of digestion, already interfered with by the portal blockage, is made still more difficult.

In the same way the spleen, the pancreas, the intestines, the liver itself, all suffer from the pressure of fluid upon them, and upon the vessels of the portal system; and the vicarious flow through venæ communicantes is interfered with in like manner. The kidneys indeed, which do not discharge into the portal system, are not directly affected by the venous block there. The renal veins are, however, affected by the pressure of the effusion into the abdominal cavity; and the congestion of the kidneys, which arises in extreme ascites solely as the result of pressure, and is at once relieved by its removal, affords most significant evidence of the disastrous effect of this continued distension of the abdomen by fluid. The urine becomes scanty, high-coloured, albuminous, and sometimes contains casts. There ensues in fact a nephritis; disturbance of the renal function; a uræmia, adding systemic poisoning to the other disorders.

Although there may be difference of opinion as to the course to be pursued in order to remove the fluid, and to relieve this general oppression of the vital organs, there can be none, I think, as to the necessity for doing so by some means or other. The only question is when and how it should be done.

There is no more painful clinical picture of hopeless disease and helpless therapeutics than these cases of ascites, left unrelieved in their increasing exhaustion and distress. The question is not one of mere relief of distress, however; it is one of releasing the embarrassed vital organ from fatal pressure, of improving the general pathological condition, and making temporary recovery possible. For, as I have shown, ascites does not arise in the last hopeless stages of atrophic cirrhosis only, but now and again appears earlier, when something more that mere temporary relief may be hoped for.

The practice adopted for this purpose of old was to attempt to carry off the fluid by hydragogue purgatives and by diuretics. Now with regard to the former, it may be affirmed, without hesitation, that hard purging is, in these cases, a pernicious and fatal practice. It is in most instances absolutely ineffectual as a means of reducing the dropsy; it is most effectual in reducing the strength and nutrition of the cirrhotic patient, already enfeebled by the difficulty of drawing a minimum of aliment through the obstructed vessels. This difficulty is increased by purgation; the nutrient fluid is hurried through the alimentary canal too rapidly for absorption. The flow is set *out*wards instead of *in*wards.

The plan of giving the purgative in the early morning before food is taken mitigates, but does not neutralise, the ill effects; a large amount of nutrient material remaining from previous meals is drained away. Further, this favours the onset of diarrhœa, which is only too apt to set in spontaneously, is most difficult to control, and of evil augury.

The expression 'purged to death' is, not unfrequently, sadly appropriate to the records of cirrhotic ascites. Therefore, although a few sharp doses of aperients may be useful at the outset, when the patient is comparatively robust, free purging is a fatal device when the patient is enfeebled; and in any case is to be deprecated as a continued treatment.

With regard to diuretics, it may be said that, where the kidneys are sound, they are harmless; where diseased, injurious. They are, moreover, constantly futile. Cases are indeed recorded where the action of diuretics is credited with the cure of abdominal dropsy. I have tried them all, and, as a means of removing the copious effusions of hepatic ascites, with entire want of success. As a matter of fact, in this condition, as Murchison pointed out, 'diuretics are of little avail.' The pressure on the renal veins so interferes with the circulation through the kidneys that diuresis becomes impossible. It has been found that, whilst the most powerful diuretics have failed to excite the flow of urine in ascites, the removal of fluid by paracentesis is often followed by a copious flow even without the aid of diuretics.

In a paper published in the 'Lancet' more than twenty years ago[1] I discussed the treatment of dropsy of various forms on the plan of restricting the inflow of fluid by restricting the drink of the patient, thus compelling the body, by limiting the intake, to reappropriate some of the outflow, and hence favour absorption of effusions.

I found that in cases of renal dropsy it was unquestionably injurious, lessening the flow of urinary excretion—in cases of cardiac dropsy of doubtful service—but in hepatic ascites usually well borne, and of some value as an adjunct to other treatment. In a recent article on dropsy Dr. Dickinson cites two cases of hepatic ascites in which this method proved successful.[1]

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[1] *Lancet*, 1878.

PARACENTESIS

The only reliable plan of removing the abdominal fluid, however, is to draw it off by mechanical means. The postponement of the operation until the vital machinery is seriously embarrassed is, I am convinced, a fatal error. Why should we persist in exhausting our debilitated patient, in the vain attempt to remove fluid by drugs, through the roundabout channel of bowel and kidney? Why waste time in these futile endeavours, and allow the vital organs to become more and more engorged and injured, when the fluid can be withdrawn directly and more effectually by the simple agency of a Southey's trocar? The good effected is great, the risk, in these days of antiseptic surgery, of the smallest.

I presume that the main reason for the tardy adoption of earlier paracentesis lies in the fear of septic peritonitis; and the fact that the weight of authority against it, except as a last resource, has been overwhelming—Sir Thomas Watson, Niemeyer, Thierfelder, Aitken, Bristowe. Some objected chiefly on the ground that it was useful only for the relief of suffering—

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[1] *Allbutt's System of Medicine*, vol. v. p. 690.

Frerichs objected to it on the ground that the counter-pressure of the ascitic fluid upon the peritoneal vessels lessened the rapidity of effusion. Others, again, objected on account of the loss of albumen involved. Murchison and Bristowe, however, modified their views at a later period, and sanctioned paracentesis before an extreme stage was reached.

I have known only four instances in which paracentesis proved fatal from resultant peritonitis. Two were in the period before antiseptic precautions were observed, the third was due to accidental displacement and infection of the drainage tube by the patient during the process ; the remaining one was unexplained.

It is true that, when the operation is postponed until the patient is in the last stage of cachexia and exhaustion, tapping sometimes hastens the final failure ; but I have never seen this in the earlier stages, before the general health has been gravely impaired.

As a matter of curious interest I may say I have recently had under my care at St. Mary's a man of forty-nine who was tapped for ascites in St. George's Hospital, I think, by Mr. Cæsar Hawkins, forty years ago.

The first physician to insist upon the advisability of tapping early was, I think, Dr. Austin Flint, in America, as long ago as 1863. Writing in 1883[1] he says: 'In concluding my clinical report on ascites twenty years ago, I used the following language—"Unpromising as are the majority of cases of ascites, I cannot but believe that, as regards prolongation of life, and as much improvement as is compatible with existing structural disease, the success of medical practice would be enhanced by employing less, than has been the custom, diuretics, hydragogue cathartics, and depressing remedies, and by resorting earlier than is usually done to tapping. After the added experience of twenty years I hold to the same belief, with a strong conviction of its correctness, as based on reason and clinical facts." ' And he gives ten cases showing favourable results in support of this opinion. In 1872 Dr. Frederick Roberts[1] advocated repeated tapping, and also gave a series of cases as evidence of its success. In 1873 Dr. McCrew,[2] and in 1881 Dr. Duncan[3] expressed similar conclusions.

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[1] *Brit. Med. Journal*, 1883, vol. ii. p. 566.

When I ventured eight years ago to urge the employment of early and repeated paracentesis in hepatic ascites, in a discussion on the subject at the meeting of the British Medical Association in Nottingham in 1892, opinion upon the subject proved to be still much divided. At that time all the text-books of medicine, as far as I know, with a solitary exception, agreed in repeating without question the old stereotyped rule, that the operation should be postponed as late as possible Now, however, this view has been modified, in so far that paracentesis is sanctioned by most authorities when the pressure of the fluid is obviously causing embarrassment of the viscera.

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[1] *Practitioner*, 1872.

[2] *Dublin Med. Journal*, 1873.

[3] *Brit. Med. Journal*, 1881.

As a matter of my personal experience, in every instance of marked hepatic ascites which has done well, early and repeated paracentesis has formed an essential part of treatment. Before this was adopted, the results, in my practice at all events, presented one long uniform list of melancholy failures; and in the vast majority of cases of hepatic ascites recorded where the condition has been successfully treated by others, repeated paracentesis has also formed a main feature of treatment.

In the least favourable form of atrophic cirrhosis even, where the good effected is only transitory, and nothing can reasonably be hoped for beyond a respite of a few months, paracentesis is beneficial, if not left too late. The early removal of the ascitic fluid enables the patient to live longer in comparative comfort, instead of struggling through a shortened life in hopeless unmitigated distress.

In discussing the question of viability and prognosis in the preceding lecture I instanced a number of cases which support this view; in most of those which proved fatal the correctness of diagnosis was proved by post-mortem examination. The only explanation of the less favourable results attained by some others, which I can offer, is that possibly the operation was postponed to too advanced a stage of disease; or not systematically repeated at proper intervals, and the case retained under close observation and treatment throughout. Tapping in the very last stage of cirrhosis frequently hastens death; and a single paracentesis without repetition, or repeated only too late when symptoms again become urgent, is of little service. In the successful cases which I have to record, the patients were retained in hospital, or under constant medical care in private, until fluid ceased to return and health was fairly restored. 'Tap and turn out' is a most unsatisfactory practice, futile and delusive.

The removal of fluid by continuous drainage has been practised with some success by Dr. Caillé and Dr. Elliot in America, and by Dr. Urso in Italy. In some cases of mine, where the puncture of paracentesis has remained open, and drainage continued for days, the results have been satisfactory, except for the manifest discomfort involved. A more drastic plan of promoting adhesions of the liver and spleen to the parietes by surgical operation has been advocated, with the idea of aiding the development of new vascular channels. It seems at first sight somewhat like the partial closure of a safety valve, preventing automatic easing of the portal vascular stress by serous oozing. Nature's operations in this direction of adhesions in cirrhosis are not apparently favourable, and associated with the most intractable cases; they do not suggest an imitation of her methods.

The course of these cases of cirrhosis which have resulted in restoration of health for lengthened periods —cases in which the return to health has been such as to warrant the term 'recovery'—is most satisfactorily illustrated perhaps by a brief description of some of the most remarkable and representative amongst them.

CASE I.—The first was that of a patient sent into St. Mary's Hospital in October 1883 as suffering from Bright's disease. He was a man of thirty, of great stature and fine build, slightly jaundiced, and he had lost a good deal of flesh. The ascites was extreme, the legs below the knees œdematous, but there was no general dropsy. The liver was greatly enlarged and hard, reaching half-way to the umbilicus, the heart displaced upwards, but the sounds normal, the lungs clear. The urine contained one-sixth albumen, and both hyaline and granular casts, but no blood.

The patient was a carman, and confessed to taking seven to eight pints of beer a day, with half a quartern of rum before breakfast, and he suffered from morning diarrhœa.

The case was regarded as one of alcoholic cirrhosis, and treated in orthodox fashion by cathartics, and diuretics, and tonics, paracentesis being postponed until urgent symptoms should arise.

The man got steadily worse, the lungs became engorged, distension extreme, dyspnœa and distress great. Then, *secundum artem*, paracentesis was performed as a mere matter of relief, 400 oz. being drawn off. The patient slept well that night for the first time for many weeks.

At this juncture the patient complained of sore throat, and a small ulcer was observed on the soft palate. It was then remembered that both the man's testicles had been removed, one for 'strumous disease' five years before, and the second eighteen months ago for 'tuberculous affection.' On further inquiry it was ascertained that he had suffered from venereal disease fifteen years ago, and chancre scars were visible. Although the condition of the liver itself was not suggestive of syphilis, it was recognised that even surgeons are human and fallible, and that, probably, the affection of the testicles was syphilitic, not tubercular. Iodide of potassium was accordingly given in doses of ten grains three times a day. This was not followed by any immediate improvement, and in three weeks the man had fallen back into his former condition of extreme distension and urgent distress.

Paracentesis was again performed, and the iodide continued. From this time improvement set in, and was continuous. Albumen disappeared from the urine. Appetite returned. Tapping was repeated four times more. The intervals between the tappings grew longer; the amount of fluid withdrawn less; and after the sixth operation the ascites returned no more.

The patient was finally discharged seven months after admission, to all appearance strong and hale, without a trace of renal or cardiac disease. The liver, however, remained large and hard as before.

The man came to hospital for examination a year and a half afterwards: he had remained well and robust, had walked ten miles with his horses the day previous. The liver condition was unchanged—enlarged and hard.

This case was especially interesting to me, because, up to this time, I had never seen recovery from hepatic ascites when the dropsy, emaciation, and exhaustion had progressed to such an extent as in this instance. In all previous cases, in spite of treatment, and I am afraid too often in consequence of treatment, the patients invariably went from bad to worse. In one or two cases I had seen ascites disappear, but never the patient get well.

The relief afforded to the oppressed organs by the removal of the ascitic fluid, the absence of any material progress under iodide of potassium until the general deadlock was relieved by a second tapping, followed by repetition, before serious visceral engorgement recurred, compelled the opinion that blind following of the routine practice of postponing mechanical relief as late as possible was a mistake, and that not only were the patients' sufferings thus needlessly aggravated, but their chance of life seriously endangered or destroyed.

Subsequent experience has amply confirmed this judgment. The absence of improvement under iodide at first, and the favourable effect of tapping, leave it uncertain how far alcohol, and how far syphilis, were factors in the case. Probably both played a part.

CASE II.—The next case is adduced as being probably an example of the condition of masked syphilis, of which I have spoken. The patient was a woman of forty, with enormous ascites, the abdomen measuring 43¾ inches. She was thin, emaciated, not cachectic, and not jaundiced. The liver was just felt below the margin of the ribs after tapping. The body had begun to swell nearly two years before, and she had already been tapped five times. She had had much pain in back and side, and five months ago her legs began to swell. There was no sign of heart or renal disease. There was no history of alcoholism, and the woman had not the appearance of a drinker. There was no history of syphilis, nor sign of it. The patient was a married woman with one healthy child and had had no miscarriages.

Physical examination showed that the dropsy was not ovarian; that there was no matting of intestines, or omentum, suggestive of tuberculosis. Alcoholism, heart disease, peritonitis due to kidney disease, or to tuberculosis, seemed all precluded—and it was judged to be most probable that the affection was syphilitic cirrhosis with perihepatitis; a condition so often neither indicated by history nor betrayed by physical evidence.

This diagnosis was strongly confirmed by the subsequent progress of the case. Pleurisy, pericardial friction, slight hæmoptysis occurred, suggesting the presence of gummata in the lung—a condition found to exist in another case of syphilitic cirrhosis.

She was treated by iodide of potassium, tapped three times more, and discharged, four months after admission, plump and well, free from all signs of cardiac, pulmonary, or renal disease; the urine, 1022, free from albumen or casts.

Five months later she came back for examination, and was found to all appearance in robust health, without sign of any dropsy, kidney disease, or heart disease, the liver being felt as before just below the ribs.

Case III.—The next case shows the extended course of syphilitic cirrhosis in some instances, its first stage resembling that of an alcoholic atrophic liver. A woman of forty-six was admitted to St. Mary's in 1885 for disease of the liver, supposed to be malignant; great ascites and marked jaundice, which had lasted for six months; and rapid loss of flesh. The liver was greatly enlarged, hard, and somewhat uneven of surface, and it was tender. She was tapped, and given iodide of potassium, and went out apparently well; came in again the following year, was treated in like manner, and again went out in good general health.

Two years later she was admitted for the third time, was again treated by paracentesis and iodide, and went out well.

She remained under observation and free from ascites for five years more, when she had an attack of hæmatemesis; a year later the hæmatemesis returned, from which, and intercurrent erysipelas, she died. No post-mortem examination could be obtained, but the liver remained greatly enlarged and hard to the last.

Case IV.—The next case which I have to bring forward is one in which the affection was purely alcoholic in origin. The patient, a publican, whom I saw in consultation on November 5, 1890, was a tall, powerfully built man: he had lost flesh, but was still somewhat stout, and not cachectic in appearance.

His abdomen was distended with fluid, measuring forty inches in circumference. The superficial veins were full and conspicuous. The liver was greatly enlarged, the edge reaching to within three inches of the umbilicus, its surface smooth—very hard—and slightly tender; the legs were œdematous.

The heart was not appreciably enlarged, the sounds regular, and free from murmur. The lungs were normal, except for some bubbling râles at both bases. The urine was scanty, high-coloured, and contained one-quarter albumen. No casts or blood could be found on microscopic examination.

The account given by the patient was that he had noticed his abdomen steadily increasing in size for about six months; his legs had only begun to swell during the last fortnight. He had never had rheumatic fever or any other serious illness. He had never had syphilis, nor was there anything suggestive of it. He attributed his illness to working in a damp cellar; and the cellar was no doubt a factor. He acknowledged to drinking enormous quantities of beer daily—spirits only on occasion.

It was agreed that paracentesis should be performed, and repeated as required. This plan was duly carried out. The distension usually returned again, so as to cause discomfort, in two to three weeks. Paracentesis was performed nineteen times in all between November and the following July—a period of upwards of eight months—amounts varying from 580 to 220 oz. being withdrawn. Iodide of potassium was given for some weeks as a measure of precaution—but without good effect, and it was abandoned, and grey powder substituted for a time. No drastic purges were given, the bowels being simply regulated by a mineral water. The albumen rapidly disappeared from the urine, after the first tapping, returning fitfully, from time to time, for short periods, with the increased pressure of reaccumulating fluid. The urine remained scanty—about 20 oz. only.

Shortly after the eighteenth tapping, however, the patient, who had previously been lying on his back on bed and couch, got up and took a short drive and walk, to record his vote at an election. On his return home—*post hoc* or *propter hoc*—diuresis set in, and continued for some days, to the amount of 100 to 120 oz. in the twenty-four hours. The fluid in the abdomen did not altogether disappear, and a nineteenth and last tapping was performed. After this there was no return of the ascites. The man's health continued to improve, and when he called upon me a year after my first visit to him he was apparently hearty and well, except that the liver was large and hard as before. There was no sign of heart disease, the urine was normal. He was able to resume an active life—he had remained a rigid abstainer.

From that time I lost sight of him, but his medical attendant, to whom I am indebted for most of the details of the case, tells me that the patient remained well, leading an active life, until about two years ago—a period of eight years in all—when he fell a victim to influenza pneumonia. From the commencement of the first illness until his death, he had entirely abstained from alcohol.

Case V.—The next example of hypertrophic cirrhosis of purely alcoholic origin, with extreme ascites, is perhaps the most satisfactory of all, since the subject survives to this day—nearly twelve years after first coming under treatment. In this instance the patient was a married woman of thirty-two, admitted into St. Mary's Hospital for jaundice and ascites, in May 1888.

She acknowledged to being a heavy drinker—of spirits chiefly—and on one occasion gave material evidence of it by coming to the hospital in a state of riotous intoxication. There was no history of syphilis, or suspicion of it. The woman was fairly nourished—not thin nor cachectic—although she stated that she had lost much flesh. Her face was covered with acne rosacea and venous stigmata, and she was jaundiced; the abdomen was greatly distended. The superficial veins were full and tortuous; the liver enlarged, reaching four inches below the ribs, extremely hard, smooth, or slightly granular. The heart-sounds were normal, the urine free from albumen. There was some œdema of the ankles.

The patient was tapped seven times in the course of the five months for which she remained in hospital, large quantities of fluid being drawn off. Iodide of potassium was given for a few weeks, but discontinued, since no improvement followed, and iron and digitalis substituted. After the seventh tapping the dropsy did not return; and the woman went out at the end of five months apparently well, and so remained for five years. She, however, took to drink again, and was admitted for the second time, in October 1893, in condition of an enormous ascites. The abdomen measured fifty and a half inches, the liver was large and hard as before, distinctly felt seven and a half inches below the costal margin, as far as the umbilicus, the hepatic dulness commencing above the sixth rib.

The heart was normal; the urine free from albumen.

She was tapped twice—386 oz.—and treated by iodide of potassium, for a short time, and then by diuretics. The latter failed to promote diuresis even after tapping. The ascites did not return, and she went out well in January 1894, three months after admission.

She was admitted for the third time in 1895, tapped several times, and then discharged well. Admitted for a fourth time in December 1896, and again went out relieved by tapping. She was admitted for the fifth time December 1897, tapped and relieved, but, on account of the enormously distended flaccid abdomen, all the fluid could not be removed. She was supplied with an abdominal belt and directed to return.

She failed to reappear, but was sought out and induced to come into hospital again in December last to complete the cure of her ascites, and this further tapping has been successfully done. She had remained in good health since her last visit two years ago. The following is her condition as ascertained December 1899—three months ago. The patient looks fat and well, except for some marking of the skin of the face from dilated vessels. The abdomen is very large, flaccid, and pendulous from previous distension, measuring forty-three inches. It contains a considerable amount of fluid which the patient says has remained since the last incomplete tapping two years ago. The liver is large and very hard, with a sharp edge six inches below the ribs; it is therefore somewhat smaller than it was two years ago. The spleen is apparently not enlarged; the superficial veins of the abdomen conspicuous, but not distended. There is no œdema of the ankles; the heart is not enlarged, there are no bruits, the sounds and action normal. The urine is acid lithatic, sp. gr. 1027, free from albumen and casts.

The fæces are normal in appearance, and show a good amount of biliary colouring.

The permanence of the liver enlargement, the preservation of nutrition, the absence of all signs of renal disease, or of heart disease, are notable. The case remains as an example of hypertrophic cirrhosis, due to alcohol, uncomplicated by any other disorder; the patient surviving in fair health up to the present time —twelve years since cirrhosis and ascites were first recognised.

I may quote another example of alcoholic hypertrophic cirrhosis with ascites which has for two years past followed a favourable course.

The patient, a gentleman of forty-seven, was referred to me as suffering from Bright's disease and ascites. He was a broad-set, burly man, full-fleshed, his face turgid, and he had some dyspnœa. The abdomen was highly distended with fluid, the veins of the surface being swollen and conspicuous. The liver was of great size, with flat, hard, wood-like surface, and sharp, unyielding edge, extending four inches below the margin of the ribs; the legs and thighs were œdematous, but there was no general anasarca, œdema of loins, or puffiness of face.

The heart was somewhat enlarged, the apex beat just outside the nipple line, and the area of deep cardiac dulness increased laterally to the right margin of the sternum. Its action was quiet and regular, the first sound somewhat short, the impulse not increased. A mitral bruit could be heard just over the apex. The pulse was 80, soft, regular, and of rather low tension. The lungs were clear, except for slight basic catarrh. The urine was diminished in quantity, high-coloured, and very acid; sp. gr. 1024; contained albumen estimated at one-fiftieth. Careful microscopic examination revealed neither blood nor casts.

The patient had indulged in alcohol habitually to great excess, up to the commencement of his illness; having, as he told me, taken sometimes as many as twenty-four glasses of whisky and soda, or more, in a day. For the last eighteen months, however, he had been a total abstainer. There was no history or sign of syphilis.

The condition was clearly hepatic ascites, due to alcoholic cirrhosis, with some dilatation of the heart, and renal congestion, dependent upon the cardiac and hepatic lesions. In view of the great abdominal pressure and distress, I advised that paracentesis should be performed without delay. This was carried out by his medical attendant in the country, and 300 oz. drawn off. The leakage continued for some time afterwards. Saline laxatives and digitalis were given internally.

The effect of the removal of the fluid was remarkable. When I next saw the patient, four weeks afterwards, the abdomen was lax, but still contained some fluid; the liver hard, and large as before, reaching to within one inch of the umbilicus. The œdema of the legs had almost gone. The area of heart dulness reached to mid-sternum. The urine was free from albumen, of sp. gr. 1021. He was already so active and free from dyspnœa, and so well, that he had been walking about the streets, and paid a visit to one of the public offices on business. His appetite was excellent, indeed exceptional, and he expressed himself as feeling perfectly well but for his remaining ascites. To sum up, he was subsequently tapped (four times more), the last time in August 1899. Since then the ascites has not returned, and the medical man under whose care he has been reports, in January of this year, that he remains active and well in all respects; the enlargement of the liver, and the slight, local, mitral bruit being the only marks of disorder. His weight is now upwards of fourteen stones.

Such is the course of the case since it came under my personal observation.

The previous history, for which I am indebted to the kindness of the medical men under whose care the patient came earlier, is of great interest, since it throws light upon the relation of the cardiac, hepatic, and renal disorders to the present condition of a more simple hepatic ascites.

In November 1897, a little more than a year before I first saw him, the patient came under the care of his medical adviser in the country in a critical condition, with great ascites, anasarca, albuminous urine, irregular pulse, feeble failing heart. He was tapped, and given digitalis, and he shortly recovered sufficiently to resume his duties away. He quickly broke down again, however, and in August 1898 came under the care of an eminent practitioner, who has kindly sent me the following account of his condition at that juncture:

'There was a history of kidney disease, enormous anasarca, and ascites. The heart was greatly dilated, with a mitral bruit, irregularity of action, reduplication of first sound, and venous pulsation; there were marked symptoms of uræmia. The urine had a sp. gr. of 1024, was highly albuminous, containing oily and epithelial casts. The liver was greatly enlarged. He was tapped nine times, to the extent of eighteen gallons and three pints, between August 1898 and January 1899. Various drugs were given; digitalis was ill borne; but strophanthus, strychnia, and salines were distinctly beneficial.'

It seems clear that in the first instance there was great cardiac dilatation, and a congestive nephritis in addition to the cirrhosis and engorgement of the liver, the cardiac dilatation being due to the enfeeblement of the cardiac wall from alcoholism. The enlargement of the liver was due, partly to alcoholic fibrosis, partly to engorgement from the heart state; whilst alcoholic irritation, cardiac inability, and ascitic pressure were probably factors all engaged in the production of the congestive nephritis. With rest, abstention from alcohol, cardiac tonics, and the removal of external fluid pressure, all the disorders dependent solely upon temporary and removable conditions subsided, the heart contracted, the nephritis ceased, the general dropsy disappeared. The condition of the liver, however, depending, not only upon venous engorgement, but upon fibrosis also, could not be so remedied. The cirrhosis remained, and the ascites returned from time to time, until the development of vicarious circulation and the increased cardiac vigour eased the portal tension.

That the course of events followed closely on these lines is shown, I think, by the immediate and great relief afforded by paracentesis, evidenced by the cessation of dyspnœa, rapid subsidence of œdema of the legs, disappearance of albumen from the urine and of all signs of renal disorder. The dropsy in the last stage was not a general dropsy, due to cardiac and renal disease, but an hepatic ascites.

The course of the disease in the preceding case suggests that cardiac inefficiency is sometimes an important agent in determining the occurrence of portal dropsy in cirrhosis.

I have now under my care at St. Mary's a man with extensive mitral disease and secondary tricuspid disease, and an enormously enlarged pulsating liver, who has very extensive ascites, but hardly any general dropsy.

In speaking of the intercurrent ascites of mitral and tricuspid disease of the heart in the first lecture, I suggested that it might be due to the engorged condition of the liver, plus the moderate cirrhosis which follows on prolonged venous hyperæmia; the addition of systemic obstruction to that of the fibrosis and capillary dilatation causing greater intravascular pressure in the portal veins than in the systemic, determines transudation in the portal area—a portal dropsy only, not a general systemic dropsy.

Evidence was adduced from morbid anatomy proving how gravely the muscular structure of the heart is often affected by degenerative changes in cirrhosis.

It is, I think, probable that in these cases of hypertrophic affection, when ascites supervenes earlier than the fibrosis of the liver alone seems to warrant, the increased portal stagnation induced by heart dilatation and inefficiency, added to the cirrhotic obstruction, may be the last straw—the determining factor of peritoneal effusion.

Two other cases which have come under my observation lend support to this view. In 1894 I was asked to see a young fellow of thirty-two, a man in wealthy permanently disappear, as it does in the analogous condition of the ascites of mitral dropsy.

There is another point, connected with this relation of cardiac inability and hepatic stagnation, which deserves consideration. Is it not possible that this may explain in some degree, not only why in these cases of hypertrophic cirrhosis the liver remains large, but also why the fibrosis affects partly the finer branches of the interstitial tree—instead of the large trunks only as usual in atrophic cirrhosis? The character of the fibrosis is certainly not determined by the nature of the alcoholic stimulant, as has been suggested—according as it is beer or spirits. It may be partly determined by degree of dilution—or concentration—of the alcoholic poison. In three of the four cases related, the patients were spirit drinkers—one only a beer drinker.

The enlargement, moreover, is not due to fatty deposit. My observations agree with those of Dr. Foxwell,[1] that fatty infiltration is no more common in the large than in the small livers. The enlargement is probably due in part to chronic venous engorgement, and dilatation of hepatic venules and capillaries, such as is found in the nutmeg liver of cardiac cirrhosis. If the microscopic specimens of pure cardiac cirrhosis are examined, and compared with that of alcoholic hypertrophic cirrhosis, it will be seen that the two conditions are almost absolutely identical. There is the same mixed coarse and fine fibrosis, the same appearance of fresh formation of biliary ducts, the same enlargement. Moreover, it might well be

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[1] *Enlarged Cirrhotic Liver*, Birmingham, 1896, p. 20.

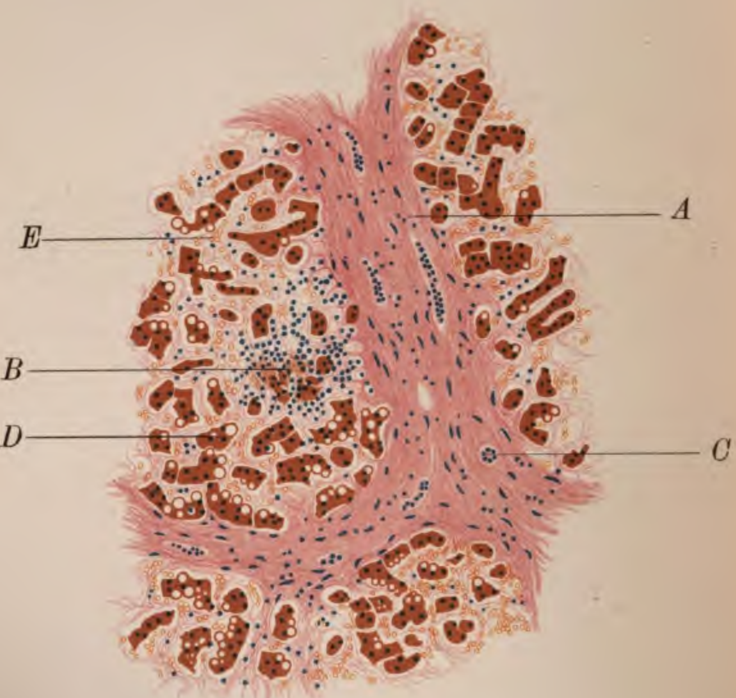

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PLATE X

CIRRHOSIS OF THE LIVER, DUE TO PROLONGED VENOUS CONGESTION FROM TRICUSPID INCOMPETENCE

The patient, a boy aged nine, was admitted for cyanosis, ascites, and breathlessness.

He had an enlarged hard liver and large spleen.

After a prolonged illness he finally died of acute tuberculosis.

Post-mortem.

The pericardium was generally adherent, and contained calcareous plates. The tricuspid ring was rigid from chronic tubercular inflammation, and in parts of the ring there was caseous deposit. The liver was cirrhotic, and there was no evidence of tubercle in the substance.

Explanation of Plate.

A. Coarse fibrosis.
B. Active cellular infiltration.
C. Pseudo-duct.
D. Liver-cells (fatty).
E. Blood.

PLATE XI

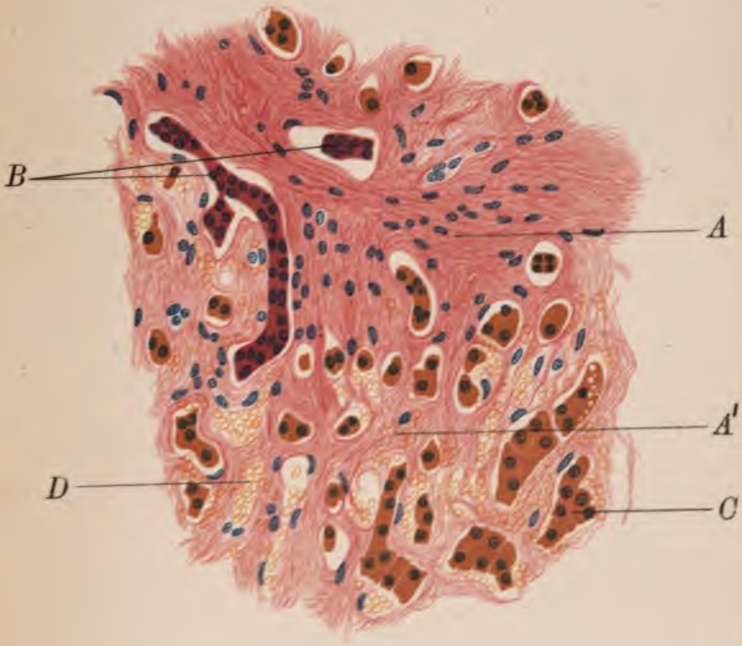
CIRRHOSIS OF THE LIVER, DUE TO PROLONGED VENOUS CONGESTION

(From the same case as Plate X under higher magnification)

Explanation of Plate.

A. Coarse fibrosis.
A′. Fine fibrosis.
B. Pseudo-ducts.
C. Liver substance.
D. Blood.

Plate XI.



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that in alcoholic cirrhosis with early enfeebled heart the effect of the alcoholic poison would be especially felt where the blood stream is most delayed, as the region of the hepatic vein in the lobule is approached, producing a fine fibrosis there; and that this region would escape when the heart factor did not come in, and a simple coarse cirrhosis only, with atrophy, in that case, result.

Be this how it may, however, I believe that cardiac inefficiency, even when not advanced enough to disclose itself by the usual physical signs of heart disease, or a general dropsy, is a factor in the causation of the more acute cirrhosis of the enlarged alcoholic liver, and of the ascites which may accompany it.

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